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THE
LARYNGOSCOPE.

VOL. LIII NOVEMBER, 1943. No. 11

SURGICAL APPROACH FOR ARYTENOIDECTOMY IN BILATERAL ABDUCTOR PARALYSIS OF THE LARYNX.

DR. HENRY BOYLAN ORTON, Newark.

The purpose of this paper is to emphasize a method of extralaryngeal approach to the arytenoid cartilage in performing arytenoidectomy for the relief of bilateral abductor paralysis. It must be remembered that the contraction of any scar tissue will be laterally and not mesially in the larynx.

This method, "The Lateral Transthyroid or Trotter's[1] Approach to the Laryngopharyngeal Aponeurosis," was mentioned in January, 1943,[2] as a simple method for performing arytenoidectomy with ample room, under direct vision.

It is a method of access; by removing the ala of the thyroid cartilage, the wall of the laryngopharynx is disclosed unopened; palpation can determine with a fair degree of precision the underlying conditions. From an anatomical and surgical standpoint, this operation gives an excellent exposure to the side of the neck. The operation in its most radical form was described in 1930,[3] and the more simplified procedure was published in 1942.[4]

King,[5] Kelly[6] Loré,[7] Hoover,[8] Chevalier Jackson,[9] Frazier[10] and others have all contributed to the relief of bilateral abductor paralysis. McCall and Gardiner[11] have recently reported a simplified operation for bilateral abductor paralysis.

King[5] says, "When I first began to experiment with the problem. it was on the theory that a sound muscle might be transposed to the arytenoid cartilage and subsequently develop a capacity to replace the function of the cricoarytenoideus

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posticus muscle. In cases of long-standing bilateral paralysis, some new problems were encountered. So the operation that was conceived as a method of opening the cords by the use of an extraneous muscle in actuality developed into a new method of cord displacement."

Kelly makes a transverse skin incision and likewise severs transversally the thyrohyoid and sternohyoid muscles; he

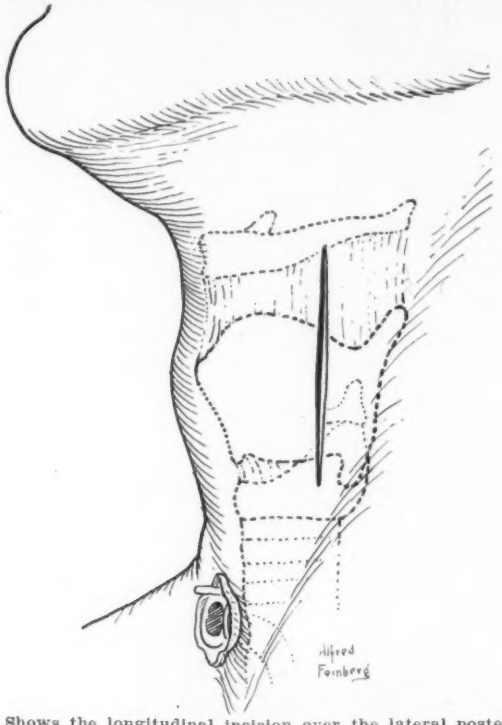

Fig. 1. Shows the longitudinal incision over the lateral posterior third of the thyroid ala.

then makes a window in the lower portion of the thyroid cartilage approximately over the arytenoid to be removed, and completes the arytenoidectomy through this window.

King, Kelly and Hoover have all reported very satisfactory results following their own procedures.

McCall and Gardiner[11] "offer their simplification in the belief that it will be helpful to those doing surgery for the

relief of bilateral abductor paralysis." They state "that the operations should be done by a two-man team, one of whom should be a trained laryngologist, familiar with the use of the laryngoscope and the direct intralaryngeal manipulation. The identification and fixation of the arytenoid cartilage is

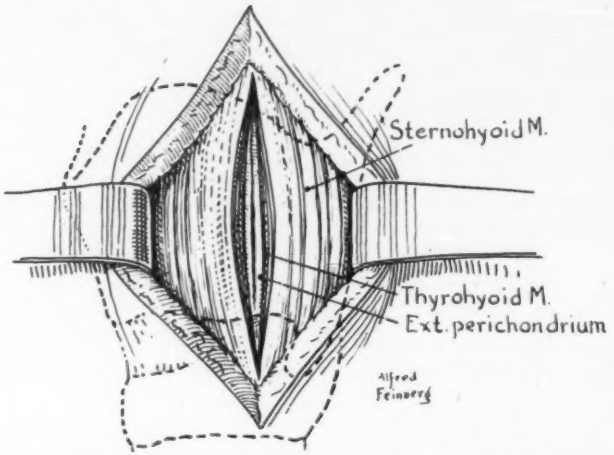

Fig. 2. Splitting of the thyrohyoid muscles and incision of the external periochondrium in line of incision.

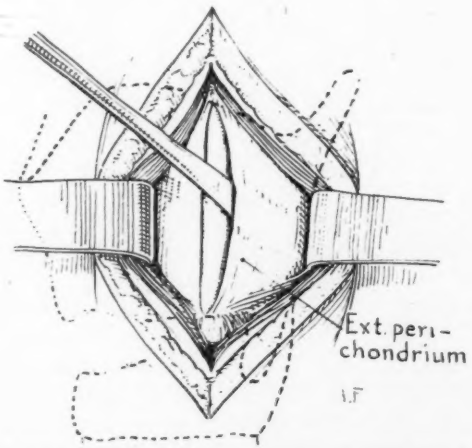

Fig. 3. Elevation of the external perichondrium from the posterior third of the thyroid ala. This separation is carried around the under-surface of same.

the Alpha and Omega of the procedure. Anyone who has done this operation realizes the greatly varying relationship between the arytenoid cartilage and the outer surface of the thyroid alae in different larynges. Just where to make the window in the thyroid alae to expose the arytenoid cartilage is not easy." Admitting this difficulty, they resort to the use of a laryngoscope which engages the arytenoid and firmly presses the arytenoid laterally against the thyroid alae. Then the window in the thyroid cartilage is made and the procedure of Kelly is carried out minus the arytenoidectomy.

LATERAL TRANSTHYROID APPROACH (TROTTER'S).

Using avertin-ether anesthesia, the position of the patient is the same as for a complete block dissection of the neck. The

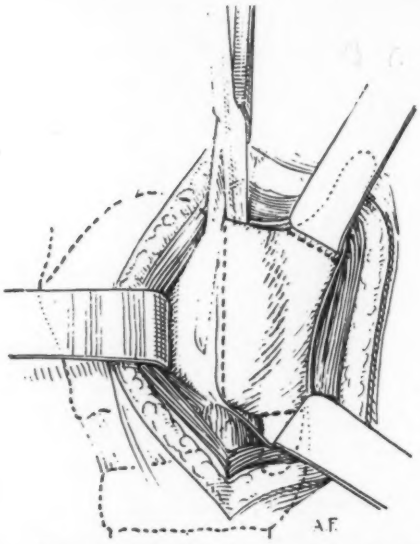

Fig. 4. Shows the perichondrium entirely separated and the posterior portion of the thyroid ala being removed by heavy shears.

chin is rotated laterally and a small sand bag or pillow is placed under the shoulder on the side of operation; this position gives a flat surface on that side of the neck. After proper preparation of the surgical field, a longitudinal incision is made over the lateral posterior third of the thyroid ala,

extending downward from a point midway between the hyoid bone and thyroid cartilage to the level of the cricoid cartilage. The sternohyoid and thyrohyoid muscles and the external perichondrium are split in the line of incision. With retrac-

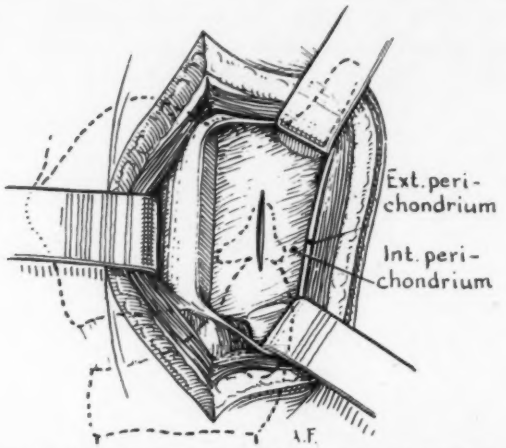

Fig. 5. Shows the internal perichondrium and the pharyngeal aponeurosis, and by palpation the arytenoid can be definitely identified, and also shows the incision over the arytenoid through its muscular attachments.

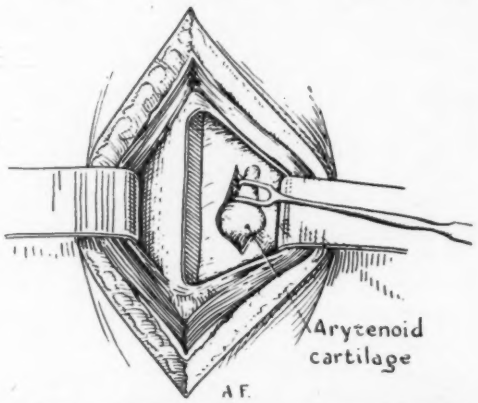

Fig. 6. Shows the arytenoid cartilage being removed from its muscular attachments.

tion laterally and mesially, the perichondrium, with the attachments of the inferior constrictor muscle, is elevated

from the posterior third of thyroid cartilage. This separation is carried around the under-surface of the thyroid cartilage with complete separation of the thyroid cartilage from the external and internal perichondrium and the posterior third of the thyroid ala is then removed by means of heavy scissors. Removal of this portion of the thyroid cartilage exposes the laryngopharyngeal aponeurosis, and then by palpation at the lower angle of the wound the arytenoid is located. Incision is then made through the internal perichondrium on to the arytenoid cartilage, and then by sharp dissection the entire

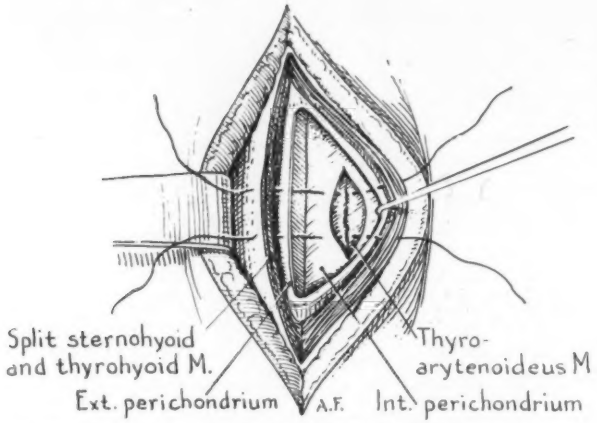

Fig. 7. Shows the insertion of sutures, which has the tendency of pulling outward that side of the larynx.

arytenoid cartilage is removed. Two fine chromic catgut sutures fix the internal perichondrium and thyroarytenoideus muscle to the external perichondrium and the anterior portion of the split sternohyoid and thyrohyoid muscles. A small cigarette drain is inserted, the fascia closed by two or three interrupted sutures and skin incision closed. This cigarette drain is removed in three to four days. No feed tube is used.

REPORT OF CASES.

Case 1: Bilateral abductor paralysis resulting from virulent laryngotracheitis following an explosion. W. H., male, white, age 18 years, suffered severe burns at the time of the powder plant explosion in 1940. Inhalation of excessively hot air caused a virulent laryngotracheitis. About three months following the explosion, Oct. 26, 1940, he suddenly developed marked laryngeal stridor which necessitated placing him in an oxygen tent. Examination on Oct. 29, 1940, revealed a bilateral abductor

paralysis with edema of the epiglottis and both arytenoids and a large amount of creamy yellow secretion being coughed up from the trachea. Under local anesthesia at the Dover General Hospital a tracheotomy was done and a No. 6 Jackson tracheotomy tube inserted. The operation and postoperative course were uneventful. Dec. 1, 1940, the swelling of the epiglottis and arytenoids had entirely disappeared, but the bilateral abductor paralysis persisted. June 20, 1941, indirect laryngeal examination revealed some movement of the right arytenoid, and the left cord paralyzed; this condition remained unchanged until Dec. 29, 1942, more than two years after the accident. Having allowed sufficient time for any regeneration of nerves, it was decided to do a left arytenoidectomy to increase the airway and allow decannulation. On Feb. 4, 1943, at the Presbyterian Hospital in Newark, under avertin-ether anesthesia, incision was made over the posterior portion of the thyroid cartilage on the left side, the incision extending from a little above the upper margin of the thyroid cartilage downward to the cricoid. The superficial fascia, the sternohyoid and thyrohyoid muscles were split in line of incision, and retracted outward. The external perichondrium was incised in line of incision and the posterior third was elevated from the thyroid cartilage with the attachments of the inferior constrictor muscle. This separation was continued on the under-surface of the thyroid cartilage and when the entire posterior third of the thyroid ala was separated it was removed in the line of incision by means of heavy scissors. On palpation, at the lower angle of the wound, the arytenoid cartilage was felt; then by sharp dissection the entire left arytenoid was freed from its muscular attachments and removed. Fine chromic catgut sutures were then passed through the thyroarytenoideus muscles and internal perichondrium and sutured to the external perichondrium and the anterior portion of the split sternohyoid and thyrohyoid muscles. A small cigarette drain was inserted and the fascia and skin closed. The drain was removed in four days. No feeding tube was necessary. Feb. 23, 1943, laryngeal examination showed a very definitely increased airway. March 1, 1943. I presented this patient before the New York Laryngological Society. The patient was decannulated on March 22, 1943. At the present time the patient has no difficulty in breathing and has an excellent voice.

Case 2: Bilateral abductor paralysis resulting from the use of a Levin tube over a period of time. J. O'C., male, white, age 40 years, was admitted to the Newark City Hospital, Dec. 5, 1928. He had been wearing a Levin tube for sometime and had developed a bilateral abductor paralysis; however, there seemed to be a very slight movement of his left arytenoid. A tracheotomy was recommended at this time but he refused operation until one week later, when the tracheotomy was performed. During the year 1929 numerous laryngoscopic bouginage were done without any relief. November, 1930, a right ventriculocordectomy was performed with no beneficial result. August, 1931, a bivalve Tucker tracheotomy tube was used, replacing the Jackson tube. In 1932, the laryngoscopic examination showed some slight movement of the left arytenoid, but the patient was unable to get along without the use of the tracheotomy tube. Sept. 27, 1933, Hoover's operation was done and the right cord without any relief. From 1928 to 1940 the patient had been wearing a trachetomy tube and was becoming rather discouraged and despondent. At this time the King operation was suggested, but he refused to have it done.

I did not see the patient from 1940 until 1943 when he consulted me and stated that numerous positions had been offered him, but on presenting himself for an interview the tracheotomy tube proved to be a definite handicap, and he wanted to know if something could be done to enable him to discard his tube and obtain employment. On July 9, 1943, a right arytenoidectomy was done, as already described in Case 1. The patient was discharged from the hospital on July 24, 1943, and at the present time his larynx shows improvement, but I feel quite certain that his left side will require operation.

SUMMARY.

The advantages of the procedure herein described are:

1. The lateral transthyroid approach gives a wider field of operation.

2. The longitudinal incision and splitting the sternohyoid and thyrohyoid instead of the transverse incision of skin and muscles.

3. Identification of the arytenoid cartilage by palpation beneath the laryngopharyngeal aponeurosis.

4. The arytenoid is under complete vision during the entire arytenoidectomy.

5. It eliminates the use of the laryngoscope.

6. It eliminates the uncertainty or difficulty as to where to make the window in the thyroid cartilage.

7. A direct extralaryngeal surgical attack to the side of the larynx.

8. Elimination of the feeding tube.

BIBLIOGRAPHY.

1. TROTTER, SIR W.: Purvis Oration on Surgery of Malignant Disease of the Pharynx. *Brit. Med. Jour.*, 1:269, Feb. 13, 1926. A Method of Lateral Pharyngotomy for Exposure of Large Growth in the Epilaryngeal Region. *Jour. Otol., Rhinol. and Laryngol.*, 35:289, Oct., 1920.

2. ORTON, H. B.: Discussion of paper by Dr. Joseph D. Kelley, "A Supplemental Report on Extralaryngeal Arytenoidectomy as a Relief for Bilateral Abductor Muscular Paralysis of the Larynx." *Trans. Am. Laryngol., Rhinol. and Otol. Soc., East. Sec.*, 1943. In press.

3. ORTON, H. B: Lateral Transthyroid Pharyngotomy. *Arch. Otolaryngol.*, 12:320, Sept., 1930.

4. ORTON, H. B.: Lateral Transthyroid Pharyngotomy. *Ann. Otol., Rhinol. and Laryngol.*, 51:774, Sept., 1942.

5. KING, B. T.: A New and Function Restoring Operation for Bilateral Cord Paralysis: Preliminary Report. *Jour. A. M. A.*, 112:814, March 4, 1931.

KING, B. T.: Abductor Cord Paralysis. *Jour. A. M. A.*, 112:9:814-823, March 4, 1939.

6. KELLY, JOSEPH D.: Surgical Treatment of Bilateral Paralysis of the Abductor Muscles of the Larynx. *Trans. Am. Acad. Ophthal. and Otolaryngol.*, pp. 133-143, March, 1941.

7. LORE, J. M.: Suggested Operative Procedure for the Relief of Stenosis in Double Abductor Paralysis: Anatomic Study. *Ann. Otol., Rhinol. and Laryngol.*, 45:679, Sept., 1936.

8. HOOVER, W. B.: Bilateral Abductor Paralysis Operative Treatment by Submucous Resection of the Vocal Cords. *Arch. Otolaryngol.*, 15:339, March, 1932.

9. JACKSON, CHEVALIER: Ventriculocordectomy. A New Operation for Cure of Goitrous Paralytic Laryngeal Stenosis. *Arch. Surg.*, 4:257, March, 1922.

10. FRAZIER, C. A.: Anostomosis of the Recurrent Laryngeal Nerve with the Descendens Noni. *Jour. A. M. A.*, 83:1637, Nov. 22, 1924.

11. MCCALL, J. W., and GARDINER, F. S.: A Simplified Operation for Bilateral Abductor Paralysis. THE LARYNGOSCOPE, 53:5:307-311, May, 1943.

224 Delavan Avenue.

BACTEROIDES SEPTICEMIA.
REPORT OF A CASE WITH RECOVERY.*

DR. JAMES J. WARING, DR. JOHN G. RYAN,
DR. RICHARD THOMPSON and DR. FRANK R. SPENCER,
Denver.

In the past many cases of septicemia, ushered in by a sore throat, have been regarded clinically as a streptococcic tonsillitis or pharyngitis caused by the streptococcus hemolyticus. Repeated blood cultures may be negative for streptococci, although a throat culture may be positive.

Cases of bacteroides septicemia are extremely rare and consequently the true etiology may not be found. In 1936, Lemierre[26] reported 20 cases of this disease, either of his own or his colleagues', with only two recoveries. His position as professor of bacteriology at the University of Paris gives the weight of authority to his observations and his subsequent report. He found the bacteroides funduliformis and fragilis as the Gram-negative organisms of the genus bacteroides. These germs are found in many of the natural cavities of the body, such as the mouth, pharynx, intestines and genitourinary tracts. They exist often as harmless saprophytes, but may suddenly become pathogenic and very virulent.

The disease may begin as a rather mild tonsillitis or pharyngitis, on one or both sides. By the fourth or fifth day there may be a severe chill followed by fever of 101° to 103° F., or even higher, and profuse perspiration. The sudden elevations of temperature may not occur until the eighth, tenth or even the twelfth day according to Lemierre.[26] There may be one sudden elevation of temperature once a day or oftener; occasionally less frequently than once a day, such as every other day; however, the septic fever is one of the outstanding features of the disease. The sore throat may be complicated by peritonsillar or parapharyngeal abscesses. On the other hand, thrombophlebitis of the tonsillar and pharyngeal veins, with extension to the internal jugular vein, may occur without clinical evidence of any abscess. The infection, and

*From the University of Colorado School of Medicine and Hospitals, Denver.

subsequent thrombophlebitis, takes place rather rapidly, and septicemia occurs in some cases, with a very mild tonsillitis. In other cases the tonsillitis is extremely severe.

Lemierre[26] resorted to ligation of the internal jugular vein as recommended by Mosher and others for thrombophlebitis of the internal jugular vein in one case without saving the patient's life. Lemierre[26] says the Germans used ligation of the internal jugular vein, for the thrombophlebitis, with rather indifferent results.

In all fairness to Lemierre,[26] and his very frank report of 20 cases with 18 deaths, it should be said that all of his patients were treated in 1930 without the aid of the sulfonamide drugs. Brown, Williams and Herrell,[25] at the Mayo Clinic, saved their patient by using sulfapyridine and we saved our patient with sulfadiazine.

Pneumonia with pleurisy, pulmonary abscesses, septic arthritis, abscesses of the liver and abscesses of almost any organ of the body constitute the serious and often fatal complications of bacteroides septicemia. Brown, Williams and Herrell's[25] patient had hepatitis with jaundice and possibly liver abscess. Not all the infections begin in the tonsils or pharynx as some begin in the urinary organs.

CASE REPORT.

Twenty-four-year-old white male entered Colorado General Hospital on Jan. 21, 1943, complaining of pain in the right side of the throat, difficulty in swallowing, dryness in the throat, fever, a slight cold in the head, dry hacking cough and difficulty in opening his mouth.

On Jan. 17 he gave 500 cc. of blood for a transfusion. On the evening of Jan. 19 he noticed his throat was sore, and he had difficulty in eating his dinner. On Jan. 20 he came to the admission office, where he was examined. He was advised to go home and go to bed, to force fluids, use hot gargles and take aspirin. At this time his temperature was 101°. When he entered the hospital on Jan. 21, 1943, his pain on swallowing was intense. The right side of his neck was a little swollen and very tender. He had a chill shortly after his arrival, and his temperature rose to 103.6°.

His previous history was essentially negative except that he had had many attacks of sore throat and tonsillitis.

Physical examination on admission revealed a well developed, well nourished young man. The blood pressure was 130/80, temperature 103.6°, pulse 100, respirations 22. The right tonsil, the right anterior and posterior pillar of the soft palate, and the right side of the uvula were all injected, red and swollen. A gland below the angle of the jaw on the right side was enlarged and tender. The heart was normal in size, shape and position, and there were no murmurs. The lungs were normal. Clinical impressions at this time were "streptococcic throat and

acute tonsillitis." In the morning of Jan. 22 he had a chill and his temperature rose to 104.4° F. He was seen at this time by Dr. Frank R. Spencer, Dr. L. W. Greene and Dr. John G. Ryan. His treatment was changed from sulfadiazine, which he had been getting since the previous night, to sulfanilamide. The right tonsil was painted with 10 per cent silver nitrate. On Jan. 23 sulfanilamide was reduced to 10 gr. every four hours. His temperature, which was normal in the morning, was 102° at 4:00 P. M. On Jan. 26 he had a chill in the afternoon which lasted about 20 minutes, during which time his temperature reached 104.5°. He had a second chill about midnight of the twenty-sixth and his temperature reached 105°. The morning of Jan. 27 his temperature had fallen to below normal, but in the afternoon he again had a chill and his temperature reached 104°. On Jan. 28 his temperature was again subnormal, but he had a chill in the afternoon and his temperature reached 105°. He had chills once and sometimes twice a day until Feb. 1. During the next four days his temperature still showed a marked tendency to swing, but it did not fall below 100° nor did it go above 104°. Between Feb. 4 and Feb. 8 his temperature ranged between 98.6° and 101° and fell to 99° on Feb. 9, his twentieth day in the hospital. His temperature hovered around 99° until Feb. 26, when for a few days it ranged between 100° and 101°. Thereafter it remained fairly close to normal until the first week in April when it rose again to 100°. He was discharged from the hospital on April 20, his temperature having been normal for the preceding 10 days. The total length of hospitalization was 90 days. On two or three occasions during the last few weeks in the hospital he had a recurrence of sore throat with a slight elevation of temperature. Since leaving the hospital he has been free from all symptoms. The pulse followed the temperature fairly closely, and the respirations were never elevated except during the first two weeks in the hospital.

On Jan. 27, 1943, eight days after onset, the right side of his face, the right temporal fossa and the upper and lower eyelids on the right side were swollen. There was enlargement and tenderness of the right submaxillary gland. On Jan. 29 he was given 200 mgm. hours of X-ray treatment to the right side of his face and 500 cc. of streptococcic convalescent serum intravenously. On Jan. 30, 1943, Dr. L. W. Greene incised and probed the right tonsillar region looking for a peritonsillar abscess, but none was found. On Jan. 31 the patient had another 500 cc. of streptococcic convalescent serum intravenously. His face at this time was more swollen.

On Feb. 1 a Quackenstedt test was done by Dr. Phillip Work. The initial pressure was 250 mm. With the compression of the left jugular vein there was no change. On compression of the right jugular vein, the pressure rose from 250 to 350. On compression of both jugulars the pressure rose from 250 to 500. The results of the test were difficult to interpret since all his trouble was on the right side. On Feb. 21 he had some difficulty in emptying his bladder. He was catheterized and 1,200 cc. of urine were obtained. On Feb. 2 he had a slight chemosis of the conjunctiva of the right eye. He was seen by an ophthalmologist who thought he did not have a cavernous sinus thrombosis. By Feb. 4 the swelling in the side of his face was much improved.

Sulfonamide Therapy: During the first 24 hours in hospital he received 11 gm. of sulfadiazine. From Jan. 22-27, inclusive, he received 20¾ gm. of sulfanilamide. Sulfadiazine therapy was resumed on Jan. 31, by which date he had received a total of 101 gm. of sulfadiazine.

During the first week in the hospital the patient had repeated nose bleeds. He had chills with drenching sweats which may have been partly due to aspirin. On one or two occasions during the first week he vomited. Local treatment of the throat consisted of occasional paintings with silver nitrate, hot gargles, throat irrigations and steam inhalations.

Beginning about Jan. 31 he had a nonproductive, rather irritating cough accompanied by pain in both sides of his chest due to pleurisy.

On Feb. 8 the symptoms were suggestive of right lower lobe pneumonia. This resolved with no residual lesion.

Over this area the breath sounds were feeble and tactile fremitus absent. The rales were very few after cough. The clinical impression at this time was area of consolidation at the right base, with pleurisy

LABORATORY DATA.

BLOOD

Date	RBC	Hb.	WBC	Differential
Admiss.	4,230,000	14.5 gm.	13,400	Polys. 90%.
1-29-43			37,250	Polys. 92%.
2- 1-43	3,920,000	11.5	24,100	Polys. 80%, lymphs. 19%, endothel. cells 1%.
2-10-43	3,730,000	11.0	11,600	Polys. 75%, lymphs. 20%, endothel. cells 5%, basophiles 1%.
2-10-43-3-10-43			11,000-17,800	Polys from 77% to 88%.
3- 8-43	3,090,000			
3-11-43			9,300	Polys. 63%, lymphs. 32%, endothel. 3%, basophiles 1%.
3-27-43			7,900	Polys 37%, lymphs. 63%.
4- 6-43	4,320,000	14.7	8,500—	Polys 62%, lymphs. 30%, endothel. 5%, eosinophiles 2%, basophiles 1%.

URINE

Date	Sp. Grv.	Albumin	Sugar	Acetone	
Admiss.	1030	Trace		Trace	Occasional pus cells in smear.
2- 1-43		2+		Trace	Many pus cells in smear.

BACTERIOLOGY

Date	Blood Culture	Throat Culture
1-22-43	No growth	H. Strept.
1-28-43	Gram-neg. Pleomorphic bacillus growing	
1-29-43	anaerobically, producing fetid odor.	
2- 2-43	Bact. fundiliformis	
	Subsequent cultures were negative	

and possibly a very small amount of fluid. The spleen was palpable on this date. The liver was not palpable.

On Feb. 25, 1943, he had a sudden attack of severe epigastric pain.

Summary: F. B. H., white, male, aged 24 years, acute septic throat, severe chills, and sweats, septicemia due to B. funduliformis, right cervical lymphadenopathy, right jugular venous thrombosis, palpable spleen, bilateral pleuritis, right lower lobe pneumonia or infarct, recovery.

Properties of the organism isolated in the blood cultures were as follows: All the cultures were pure cultures of a Gram-negative rod which grows only under anaerobic conditions. The blood cultures in the University of Colorado General Hospital are taken into bottles containing 150 cc. of 2 per cent tryptose containing 0.5 per cent NaCl, 0.05 per cent dextrin, 0.03 per cent dextrose, 0.3 per cent beef extract and 0.1 per cent agar. All anaerobic organisms grow readily in

the depths of this medium. The organism was very pleomorphic, sometimes occurring as very short coccobacilli, sometimes as long curved filaments frequently with club-shaped ends and often with spindle-shaped swellings. Glucose, maltose and levulose were fermented with the production of acid, but with only occasional small bubbles of gas. On anaerobic blood plates two types of growth occurred. Most frequently only very minute colonies or only a continuous film of growth without colony formation occurred. At times definite discrete colonies of about 5mm. in diameter were formed. A green discoloration of the blood was usually produced. Only occasionally true hemolysis was observed. No growth occurred on ordinary media without addition of serum. In serum or blood broth the growth was flocculent.

One rabbit injected subcutaneously with a serum broth culture of the organism died in seven days. A burrowing but partly capsulated abscess had spread from the site of injection (on the abdominal wall) down the fascial spaces of the leg. The abscess had ruptured into the peritoneal cavity which contained a very large amount of sticky, creamy pus which covered the surfaces of the liver, spleen and diaphragm. Sections of the liver showed polymorphonuclear exudation on the surface and a purulent inflammation extending some distance into the liver substance. Smears of the pus showed numerous small Gram-negative rods and anaerobic cultures yielded the original organism in pure culture. The organism was also recovered from the heart's blood. A second rabbit, which received an older culture intraperitoneally, died after 30 days and showed the same creamy exudate in the abdominal cavity.

Classification of the Organism: The properties of the organism, as described, are entirely in agreement with those described by various workers, for bacteroides funduliformis. The bacteroides are Gram-negative, nonsporulating anaerobes which are normal inhabitants of the upper respiratory, genital and intestinal tracts, which have frequently been implicated in ulcerative processes of the mucous membranes and may invade the tissues causing septicemia and abscesses. The literature on these organisms has been adequately reviewed by Dack.[34]

Because of the rarity of such cases we believe that this case will be of interest to the medical profession. We are indebted

to Dr. Edward R. Mugrage for valuable help. Not all of the articles listed in the bibliography have been quoted, but all have been read at least as an abstract by some one of the authors.

BIBLIOGRAPHY OF BACTEROIDES SEPTICEMIA.

1. Castellani and Chalmers: Manual of Tropical Medicine, 3rd Ed., New York, Wm. Wood and Co., 1919.

2. Bergey, David Hendricks: Manual of Determinative Bacteriology: A Key for the Identification of Organisms of the Class Schizomycetes, (a) 4th Ed., p. 404, 1934; (b) 5th Ed., p. 556, 1939.

3. Halle, J.: Recherches sur la Bacteriologie du Canal Genital de la Femme (etat normal et pathiologique). Travail due Laboratoire de M. le Prof. Grancher. These, Paris. 1898.

4. Veillon, A., and Zuber, A.: Zur Quelques Microbes Strictement Anaerobies et Leur Role dans la Pathologie Humaine. *Compt. Rend. Soc. de Biol.*, Paris, 10 s., IV:253-255, 1897.

5. Rist, E.: Etudes Bacteriologiques sur les Infections d'origine Otique. These, Paris, 1898.

6. Guillemot, L.: Recherches sur la Gangrene Pulmonaire. These, Paris, 1899.

7. Guillemot, L.; Hall, J., and Rist, E.: Recherches Bacteriologiques et Experimentales sur les Pleuresies Putrides. *Arch. de Med. Exper. et d'anat. Path.*, Paris, 1904, XVI:571-640, Nos. 5 and 6, Sept. and Nov., 1904.

8. Cottet, J.: Recherches Bact. sur les Suppurations Periurethrales. These, Paris, 1899.

9. Jeannin, C.: Etiologie et Pathogenie des Infections Puerperales Putrides. These (Recherches cliniques et bacteriologiques), G. Steinheil, Paris, 1902.

10. Tissie, H.: Recherches sur la Flore Intestinale Normale des Enfants Ages d'un an a Cinq. Ans. *Ann. de l'Inst. Pasteur*, Paris, 22:189-207, 1908.

11. Distaso, A.: Sur les Microbes Proteolytiques de la Flore Intestinale de l'Homme et des Animaux. *Centralbl. f. Bakteriol.*, 59:97-03, June 24, 1911.

12. Gilbert, A., and Lippmann, A.: Recherches Bacteriologiques sur les Cholecystites. *Compt. Rend. hebd. Soc. de Biol. Pan*, LIV:989-992, 1902.

13. Ghon, A., and Mucha, V.: Beitrage z. Kenntnis der Anaeroben Bakterien des Menschen, 3. Zur Aetiologie der Peritonitis. *Centralbl. f. Bakteriol., etc.*, Originale, Bd. XXXIX:497, 1905.

14. Kisskalt: Zur Pathogenistichen Bedeutung des Bacillus Funduliformis. *Deutsch. med. Wchnsch. Schrat.*, Leipz. u. Berl., XXXI:1270, 1905.

15. Runeberg, B.: Studien uber die bei Peritonealen Infektionen Appendikularen, etc. *Arb. a. d. Path. Institut. d. Univers. Helsingfors*, I:271, Berlin, 1908.

16. Heyde, M.: Ueber Infektionen mit Anaeroben Bakterien, etc. *Beitr. z. Klin. Chir.*, LXVIII:642-669, 1910.

17. Teissier, P.; Reilly, J.; Rivalier, E., and Layani, F.: Contribution a l'etude des Septicemies Primitive a Microbes Anaerobies. Etude Clinique et Experimentale d'un cas de Septicemie due au "*Bacillus Funduliformis*," Paris, *Med.*, 1:297-306, March 30, 1929.

18. TEISSIER, P.; REILLY, J.; RIVALIER, E., and STOFANESC, V.: Les Septicemies Primitives dues au "*Bacillus Funduliformis*," Etude Clinique, Bacteriologique Experimentale. *Ann. de Med.*, 30:97-144, July, 1931.

19. PHAM, HUU. CHI: Les Septicemies dues au Bacillus Funduliformis. These de Paris, 1935.

20. ROSENOW, E. C., JR., and BROWN, A. E.: Septicemia: Review of Cases, 1934-1936, Inclusive. *Proc. Staff Meet., Mayo Clin.*, 13:89, Feb. 9, 1938.

21. THOMPSON, LUTHER, and BEAVER, D. C.: Bacteremia Due to Anerobic Gram-Negative Organisms of Genus Bacteroides. *Med. Clin. N. Am.*, 15:1611-1626, May, 1932.

22. BEAVER, D. C.; HENTHORNE, J. C., and MACY, J. W.: Abscesses of the Liver Caused by Bacteroides Funduliformis. Report of Two Cases. *Arch. Path.*, 17:493-509, April, 1934.

23. DIXON, C. F., and DEUTERMAN, J. L.: Postoperative Bacteroides Infection: Report of Six Cases. *Jour. A. M. A.*, 108:181-185, Jan. 16, 1937.

24. HENTHORNE, J. C.; THOMPSON, L., and BEAVER, D. C.: Gram-Negative Bacilli of the Genus Bacteroides, *Jour. Bact.*, 31:255-274, March, 1936.

25. BROWN, A. E.; WILLIAMS, H. L., and HERRELL, W. E.: Bacteroides Septicemia. Report of Case with Recovery. *Jour. A. M. A.*, 116:402-404, Feb. 1, 1941.

26. LEMIERRE, A.: On Certain Septicemias Due to Anaerobic Organisms. *Lancet*, 1:701-703, March 28, 1936.

27. CATHALA, J.; BOURGEOIS, J., and GABRIEL, P.: Deux cas de Septicemies Primitive due au Bacillus "*Funduliformis*." *Bull. et Mem. Soc. Med des Hop. de Paris*, 49:100-103, Feb. 6, 1933.

28. LEMIERRE, R.; REILLY, J.; LAYANI, F., and FRIEDMAN, A.: Septicemie Primitive due au "Bacillus Funduliformis." *Bull. et Mem. Soc. Med. des Hop. de Paris*, 49:165-171,Feb. 13, 1933.

29 JAME, L., and JAULMES, CH.: Un cas de Septicemia a "Bacillus Funduliformis." *Bull. et Mem. Soc. Med. des Hop. de Paris*, 49:970-973, July 17, 1933.

30. HERRELL, W. E., and BROWN, A. E.: Treatment of Septicemia; Results Before and Since the Advent of Sulfonamide Compounds. *Jour. A. M. A.*, 116:179-183, Jan. 18, 1941.

31. CLAUS, H.: Uber 100 Fälle von Septico-Pyämie nach Angina. *Med. Klin.*, 27:1269, 1931.

32. KISSLING, K.: Uber Post-Anginose Sepsis. *Munch. med. Wchnschr.*, 76:1163, 1929.

33. LEMIERRE, A.: Sur an Cas de Septic Phophemie a Bacillus Funduliformis. Maladies Infestieuses, Paris, 1935.

34. DACK, G.: Nonsporeforming Anaerobic Basteria. *Bact. Reviews*, 4:227-259, 1940.

TRANS-SEPTAL SPHENOID APPROACH WITH IMPLANTATION OF RADIUM TO PITUITARY FOSSA AS TREATMENT FOR ADENOMATOUS TUMORS OF THE PITUITARY GLAND.

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The treatment of adenoma of the pituitary gland is both surgical and nonsurgical according to the best authorities. The following case histories are a report of my attempt to treat this condition by a combination of surgical approach and radiation therapy, the approach being made through the nasal cavity and accompanied by the implantation of radium in the substance of the tumor mass, being cognizant of the fact that it has been considered dangerous to use radium in this area because of the proximity of vital structures, namely, optic nerves and adjacent brain centers.

During my stay in Vienna, in 1926 and 1927 I was privileged to work with Dr. Max Hirsch. At that time he was doing a subtotal removal of the pituitary by this route. In 1934, Dr. Ridges, of Salt Lake City, reported in the *Transactions of the Pacific Coast Oto-Ophthalmological Society*, seven cases cured by the implantation of radium in the pituitary fossa. The trend of surgical judgment in the method being changed to the transfrontal approach by the neurosurgeons overshadowed the trans-septal invasion of this area to the point that all cases in this vicinity were referred to the neurosurgeons. The work of Dr. Ridges, however, stimulated my dormant idea that the trans-septal approach to the pituitary fossa was still feasible, particularly when radiation could be applied in the immediate locality of the tumor without damage to the vital structures above named, and it was through this medium of stimulation that, notwithstanding the brilliant results of the neurosurgeons and the Roentgenologists, I was prompted to treat the first patients who came to me in the manner that will herein be set forth.

It is my feeling that one who is familiar with the use of nasal illumination and instrumentation can remove as much

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of a tumor in the pituitary fossa as can be removed by a more complicated surgical procedure, namely, via the transfrontal route of the neurosurgeon.

Let it be understood that the tumors considered in this essay are those tumors of the anterior pituitary lobe of the nonmalignant type, namely, adenoma.

It was Marie who, in 1889, first called attention to the tumors of the pituitary associated with acromegaly. Babinski and Froleich, in 1900 and 1901, reported tumors of the pituitary with the different symptom complex known as adiposogenitalis. These men brought to us the two pituitary syndromes caused by adenoma of this gland in adults, that is, the anterior portion of this gland of epithelial origin derived from Rathke's pouch. The difference in these symptom complexes has been shown to be due to the predominance of different cell components. The acromegalic is associated with a predominance of acidophilic or chromophile cells, these cells being normal components of the gland that are demonstrated in a more than ordinary preponderance. The adiposogenitalis syndrome complex is associated with a predominance of chromophobe cells. The details of the above named syndrome groups are familiar to all of us and require no further comment, except to say that if the overgrowth of these particular cell groups occurs in the first named instead of the acromegalic state, we see a different although comparable situation of gigantism caused by an over secretion of the gland that is manifested before the fusion of the epiphyseal lines in the long bones.

If the tumor is of the chromophobe type, the condition is then one of hypopituitarism characterized by excessive fat and the lack of sexual development. The appearance of this symptom after puberty is a well known condition that requires no explanation at this point except to say that it is associated with increase in fat deposits and a regression toward the female hirsute with a predominance of girdle adiposity.

Although the physical manifestations changing, as they do, the appearance of patients suffering from an increase in volume of the pituitary fossa, appear long before the visual symptoms accompanying this condition, it is infrequent to find these people applying for medical help before there is at

least some loss of vision. This is always accompanied by headaches of varying degrees of severity. Perhaps it is because of the very slow and insidious onset of the physical changes that those people associating with the patient do not appreciate the change in physical contour so that almost invariably the first symptom complained of is disturbance of vision, and in the cases to be reported here, it was this symptom alone which brought the patient to seek medical aid.

The surgical approach for tumors of the pituitary fossa was first attempted by nose and throat surgeons because of the proximity of this area to the nasal cavity and the familiarity of the surgeon with the minute anatomy of the postnasal area.

In 1906, Schoffler made the first surgical exposure of the pituitary fossa by way of the nasal cavity. This was accomplished by an extensive resection of the septum, postethmoid and sphenoid sinuses. Then an attempt was made to resect the tumor.

This operative procedure was simplified by Kanavel, Halstead and Max Hirsch, of Vienna, beween 1907 and 1910. The procedure was further modified by Dr. Harvey Cushing, whose procedure differed from that of Hirsch in that the tip of the nose was freed and raised to give a direct view into the field of operation, thereby deviating from the method employed by men trained in the surgery of the nasal cavity.

During the same period external operations were being tried and devised by Krause, Fraser and Heuer. All of these operations have in common a frontal approach and today are fused into one general procedure for the approach of this particular area. It may be said that the above mentioned procedure has entirely supplanted the transnasal approach. This is to be expected as a result of the diligence and technique today employed by the neurosurgeons who, like all except the nose and throat surgeons, must have a wide area of exposure in order to perform a surgical procedure with indirect illumination.

Another treatment of the above named conditions is that of X-ray therapy. For the past 30 years X-ray treatment of the pituitary tumors has been in vogue and during this time a large number of reports has appeared and the beneficial

effects of radiation have been fully described. It has also been shown that these tumors are very radiosensitive.

Gramegna, in 1907, reported the first case treated with X-ray. His patient was an acromegalic and received temporary benefit. In about two years, Beclere reported further on a similar case that still showed improvement 13 years later.

In 1925, Garini reported four cases showing a quick response to X-ray. Also, in 1925, appeared the papers of Dott, Bailey and Cushing. It was recommended by them that X-ray should be tried and a careful check made of visual fields, unless the patient was in immediate danger of losing vision. In another paper in 1925, Bailey stated that if the vision continued to fail, surgery was indicated, but that radiation should be used after the operation.

In 1926, Hinismann and Czerny reported 15 cases in which favorable results were obtained. Kaplan also reported, in 1925, a complete return of vision in a patient with pituitary adenoma.

In 1928, Nemonaw and Jugenberg reported their results in 29 patients with headache and loss of vision, with improvement. In 1929, Sgalitzer showed that cystic adenoma of the pituitary did not respond to radiation and also little benefit was to be expected when the symptomatology of adiposogenitalis was present.

In 1930, Towne reported five cases of adenoma and his conclusions were that tumors that had enlarged and eroded the sella were amenable only to radiation. He thought that in one of his patients too prolonged treatment had caused the tumor to become cystic, causing an already improved vision to decrease.

Fraser also, in 1930, reported a case of pituitary tumor that had been benefited by Roentgen therapy but thought that good results occurred only in exceptional cases and that the optic discs and rapidity of failure of vision should determine whether X-ray therapy or surgical procedure should be employed. It was his belief that patients treated by radiation were only temporarily benefited.

Pfahler and Spackman reported three cases greatly improved by radiation and suggested that this type of treat-

ment should be used for at least six weeks before other measures were instituted.

Dyke and Hare, in reporting 63 cases, report that 48 of the 63 had X-ray without other treatment, and that 15 became progressively worse or failed to improve and were operated upon. These 15 cases have been added to the 48 treated only by X-ray, since they had fairly adequate treatment prior to operation. In all cases the diagnosis of pituitary adenoma was definitely established.

The following list of treatments of pituitary adenomas is again enumerated before describing the procedure with case reports:

Trans-septal sphenoid approach.

Transfrontal approach.

X-ray therapy.

Trans-septal sphenoid approach with the implantation of radium.

The detailed description of the last named procedure is as follows:

1. The vibrissae of both nostrils clipped short.

2. Careful application of tincture of merthiolate to the skin of the face and entrance to nostrils.

3. Application of cocaine 20 per cent, adrenalin 1-1,000, equal parts on cotton tampons in both nostrils.

4. Infiltration of columella with 1 per cent novocaine to which 10 drops of adrenalin 1-1 000 to the ounce were added.

5. Incision in the skin of columella with subsequent elevation of mucous membrane from cntire septum, followed by a removal of cartilaginous and bony portion, exposing the rostrum of sphenoid, breaking through the rostrum with exposure of both sphenoid sinuses, the mucous membrane of sinuses entirely removed, the cavity painted with applicators saturated in tincture of merthiolate. In all but one of the cases, the roof of the sphenoid sinuses was eroded through the bone and the tumor presented in the sinus cavity.

6. The presenting tumor removed by small biting forceps

and a capsule containing 50 mgm. of radium implanted in the center of the tumor mass.

7. A strip of small Penrose tubing laid between the flaps of septum to maintain drainage, both nasal cavities then lightly packed with vaseline gauze strips; a linen thread in the eye of the radium capsule allowed to protrude for a short distance onto the cheek where it was anchored with adhesive tape, serving as a means of removing the radium after the desired time had elapsed.

8. The Penrose tubing was removed with the radium and the septum flaps allowed to close, keeping the vaseline gauze packing in each nostril for another 24 hours.

In the five cases herein reported, the dose of radium was gradually increased until the third case was encountered, in which the time was greatly lengthened. The preliminary tabulation of dosage is as follows:

Case 1: 600 mgm. hours.
Case 2: 800 mgm. hours.
Case 3: 1,200 mgm. hours.
Case 4: 900 mgm. hours.
Case 5: 1,000 mgm. hours.

In no case was there any infection or undue postoperative sequelae. In all cases there was an almost immediate improvement in vision, due probably to the decompression of the tumor mass; and in all cases the improvement in vision lasted, except in the third case which ended fatally as a result of an overdose of radium that was revealed at postmortem and will be discussed in the following case reports.

Case 1: Mrs. F. S., age 43. Admitted to Holy Cross Hospital, Salt Lake City, April 9, 1937.

Chief Complaint: Severe headache, three months' duration. Failing vision for two months. Had been examined by ophthalmologists three times in the last year because of changing vision.

Family and Personal History: Born in Germany. Mother of five children, the youngest aged 3. Thought vision became worse after the birth of last child. No recent serious illnesses. Last general physical examination one month ago. Only positive finding at that time was an increase in blood pressure, 170/90. Recheck of general physical condition revealed the fact that over the last three or four years she had increased in weight, which was accompanied by a change in the facial contour together with change in size of hands and feet. Basal metabolism +6. Heart and lungs normal, blood pressure 165/90.

Examination of Eyes: External examination negative. Fundus of each

eye showed moderate arteriosclerotic change of vessels, otherwise normal. Visual fields showed a bitemporal hemianopsia for red with a segment defects for form (see perimeter charts).

Examination of Nose: Septum deviated moderately. No pus in either nostril.

X-ray Examination: Examination of the skull, lateral view, showed thinning of the anterior floor of the sella with erosion of the clinoid processes. The X-ray appearance of the sella strongly suggested an excessively large tumor of pituitary fossa.

Urinalysis: Specific gravity 1.033. Albumin 1+, sugar none, 1+ granular casts. Hemoglobin 83 per cent. Blood count: Red 4,200,000, white 7,900. Coagulation time 5½ minutes, bleeding time 1½ minutes. Wassermann negative.

This patient was admitted to Holy Cross Hospital, Salt Lake City, on April 9, 1937. On April 10, 1937, the above named trans-septal sphenoid approach was made under local anesthetic. Upon entering the sphenoid cavity, it was found that the posterior superior wall was eroded and the tumor mass presented in the sinus cavity. Part of this mass was removed and a 50 mgm. capsule of radium implanted in the center of the tumor.

Pathological report revealed a chromophile adenoma of the pituitary The radium was left in place for 600 mgm. hours. Postoperative course was uneventful, the highest temperature being 99.4°. Patient was discharged from the hospital on April 26, 1937. Postoperatively, the patient had no headache.

On May 20, 1937, the visual fields for form had returned to almost normal. There was, however, no change in the bitemporal hemianopsia for red. Uncorrected vision was 20/200 right, and 20/100 left. Corrected vision was 20/65 right, and 20/50 left.

On June 30, 1937, the hemianopsia had regressed to a segment defect, and the uncorrected vision was 20/100 right, and 20/100 left. Corrected vision was 20/50 right, and 20/50 left.

On Oct. 29, 1937, the fields for red had increased to almost within normal limits and uncorrected vision was 20/65 right, and 20/50 left. Corrected vision was 20/30 right, and 20/30 left.

This patient was followed with visual fields and determination of vision until the time of her death, which occurred on June 20, 1940, as a result of a cerebral accident that was attributed to her cardiovascular renal complex. A postmortem was obtained and a pituitary body about half the size of the sella was found. Cause of death was determined as a massive hemorrhage of the middle cerebral artery on the right side.

Case 2: Mr. W. N. R., age 56. Admitted to United States Veterans Hospital, Salt Lake City, March 30, 1938.

Chief Complaint: Severe protracted headache for six months. Gradual failure of vision for one year. Had had numerous refractions, glasses did not seem to improve vision.

Family and Personal History: Mother and father deceased, cause old age. Mother died at 77, father at 80. No previous illnesses, no operations.

Physical Examination: White adult male, well developed. Mentally clear, not acutely ill. Had large lionius facies, large hands and feet. Stated that condition of face and hands had grown progressively worse in last two or three years. Height 71 inches, weight 176. Heart slightly increased in size, no murmurs. Blood pressure 138/90.

Examination of Eyes: Vision 20/300 right, 5/200 left. External examination negative. Fundi showed no definite change. Visual fields showed a definite bitemporal hemianopsia for color and form.

X-ray Examination: Sellatursica thinned with an absence of clinoid processes. Definite increase in anterior posterior diameter.

Examination of Ears, Nose and Throat: Negative.

Urinalysis: Specific gravity 1.023. Albumin negative, sugar negative, 2,100 cc. in 24 hours. Wassermann negative. Blood count: Red 6,500,000, white 5,500. Hemoglobin 94 per cent. Coagulation time 4½ minutes. Blood chemistry nonprotein nitrogen 42. Urea nitrogen 18.6. Creatinin 1.6. Chlorides as sodium cl. 460.

On April 5, 1938, the above trans-septal procedure was performed with the implantation of a 50 mgm. capsule of radium which was left in place for 800 mgm. hours. Temperature following operation did not exceed 100°. Pulse changes were inconsequential. There was, however, a rather protracted vomiting during the time the radium was in place. There was no postoperative infection. The severe headache complained of previously to operation did not recur after operation.

The hemianopsia for color and vision gradually improved and on April 29, 1938, the visual fields defect had returned to normal for form and there was but a residual segmentary defect for color. On June 6, 1938, visual fields were taken again and found to be within normal limits for color and form. At this time, the visual acuity uncorrected was 20/100 right, and 20/200 left. Corrected vision was 20/40 right, and 20/40 left.

Case 3: Mr. J. M., age 46. Admitted to United States Veterans Hospital, Salt Lake City, April 15, 1938.

Chief Complaint: Severe headache. Progressive loss of vision, duration two years. Eight months before examination was operated upon by the transfrontal route. Because of respiratory failure after removal of the bone flap, operation was discontinued and the tumor was not removed. Following this, however, he had some alleviation of the severe headache, but there was no change in visual acuity. The headache became so severe at times that he was not able to work. It was also accompanied by nausea and some vomiting. Had had one course of X-ray therapy that seemed to increase the symptom rather than decrease it, consequently this treatment was discontinued.

Physical Examination: White male, rather well nourished, with a definite acromegalic appearance. Height 64 inches, weight 156. Heart and lungs negative.

Examination of Eyes: Vision 20/200 right, 20/200 left. Corrected vision 20/100 right, and 20/100+ left. External examination negative. Fundi negative. Visual fields revealed a complete bitemporal hemianopsia.

Examination of Nose: Moderately deviated septum with no pus, no excessive congestion in either nasal fossa.

X-ray Examination: Sellatursica revealed a marked elongation with erosion of the anterior and posterior borders. Erosion of the anterior border suggested invasion of the sphenoid sinuses.

Urinalysis: Specific gravity 1.020. Albumin none, sugar none, 2300 cc. in 24 hours, reaction acid. Wassermann negative. Blood count: Red 5,300,000, white 6,500. Hemoglobin 90 per cent. Coagulation time 5 minutes, bleeding time 1 minute.

On April 16, 1938, the usual exposure of the pituitary fossa was made under local anesthetic and the sphenoid sinus was found to be almost completely filled with a large tumor projecting from the pituitary fossa. A large piece of this was removed and a 50 mgm. capsule of radium

implanted in the remaining tumor mass and left in place for 1,200 mgm. hours. The first two postoperative days were uneventful. On the morning of the third day, patient complained of almost complete loss of vision and severe headache that did not respond to opiates. There was an increase in the respiratory rate and in the cardiac rate. This was accompanied by severe vomiting. The symptom of neurocranial pressure together with cardiac and respiratory failure increased for the next 24 hours, and the patient died on the morning of the fifth day — April 21, 1938. Pathological report of the tumor revealed it to be chromophile adenoma of the pituitary with many cells and little interstitial tissue.

Postmortem examination of the pituitary area and base of brain revealed a marked edema at the base of the brain and the remaining portion of the tumor mass in the pituitary fossa. Cause of death was attributed to an overdose of radium at the base of the brain.

Case 4: Mr. W. H. F., age 62. Admitted to the United States Veterans Hospital, Salt Lake City, on Oct. 13, 1939.

Chief Complaint: Headaches and failing vision for two and one-half years. The vision in left eye was particularly poor. Headaches were becoming more aggravated and had been accompanied by nausea.

Physical Examination: White adult male, well developed. Mentally clear, not acutely ill. Height 72 inches, weight 186.

Examination of Eyes: External examination negative. Fundi negative. Visual fields showed a marked bitemporal hemianopsia for red and a bitemporal quadrant defect for form. Vision uncorrected was 20/200 right, and 10/200 left.

Examination of Ears, Nose and Throat: Negative.

X-ray Examination: Radiographs of the sellatursica disclosed a marked elongation of the sella with an erosion of the anterior and posterior processes with a thinning out of the anterior surfaces of the fossa.

Urinalysis: Specific gravity 1.020. Albumin none, sugar none, 2,500 cc. in 24 hours, reaction acid. Wassermann negative. Blood count: Red 6,000,000, white 6,000. Hemoglobin 90 per cent. Coagulation time 4½ minutes, bleeding time 2 minutes.

On Oct. 16, 1939, the usual approach was made to the above mentioned area and a 50 mgm. capsule of radium was implanted in the tumor mass after the removal of that part which was found protruding into the sphenoid sinus. This was left in place for 900 mgm. hours. There was a moderate postoperative nausea, no headache and no infection. Pathological report showed a mixed type tumor with the chromophile cells predominating.

The visual acuity on Dec. 20, 1939, showed not much change in the character of the hemianopsia, but the visual acuity had increased to 20/100 right, and 20/30 left, uncorrected. On Jan. 25, 1940, the vision was 20/30 right, and 20/30 left, with little or no change in the character of the bitemporal hemianopsia.

These fields were taken and the visual acuity remained the same until Jan. 1, 1942, when the patient appeared and complained of a rather sudden failure in vision with a return of headache. At this time vision was 20/100 right, and 20/100 left, with a very decided change in the contour of the hemianopsia defect. He was sent to another veterans' facility for deep X-ray therapy. After a course of such therapy he returned for a further check-up, which showed the vision was 20/200 right and 20/100 left, but there was little or no field for red and a marked bitemporal hemianopsia for form. After a rest of three months, he was sent back home for more deep therapy and consequently has not been contacted since.

Case 5: Mrs. M. M., age 34. Admitted to Holy Cross Hospital, Salt Lake City, March 7, 1940.

Chief Complaint: Severe headaches for three years, last six months had been accompanied by vomiting and severe nausea. Had noticed that her shoes, hats and gloves had increased three sizes in five years. Her attending physician suggested deep X-ray therapy. She had had two courses of this treatment in the last four months. Following each treatment there had been severe nausea with some increase in headache, and after the last two X-ray exposures the vision suddenly became worse. Because of this sudden impairment of vision, she was referred to me for consultation.

Physical Examination: White female, whose features were that of a far advanced acromegalic state. Height 62 inches, weight 180. Heart and lungs negative.

Examination of Eyes: On March 6, 1940, vision was 20/50 right, and 20/50 left. External examination normal. Fundi normal. Visual fields showed a marked bitemporal hemianopsia for form and color.

X-ray Examination: Sellatursica revealed an elongation in the anterior and posterior diameter with erosion of these surfaces.

Urinalysis: Specific gravity 1.031, reaction acid. Albumin none, sugar none, casts none. Wassermann negative. Blood count: Red 5,300,000, white 7,700. Coagulation time 4½ minutes, bleeding time 2 minutes.

On March 8, 1940, following a submucous resection and the opening of the sphenoid sinus, a moderate size tumor was uncovered after removing a thin, paper-like covering that constituted the posterior superior wall of the sphenoid sinus. A moderate amount of the accessible tumor was removed and a 50 mgm. capsule of radium was introduced into the center of the tumor mass. This was allowed to stay in this position for 1,000 mgm. hours. During the time the radium was in place, there was a protracted nausea with vomiting, no headache, no rise in temperature. The immediate convalescence was uneventful. The patient was discharged from the hospital on March 15, 1940.

On April 17, 1940, following a complete recovery of the bitemporal hemianopsia defect in the left eye with a complete recovery of the visual field for form with a small superior segment for color remaining, the vision uncorrected was 20/25 right, and 20/15 left. On Nov. 12, 1940, vision was 20/25 right, and 20/15 left, with the fields essentially the same as at previous examination.

Following the examination of November, 1940, the patient moved to a nearby city and did not return to Salt Lake City until May of 1942, at which time further field studies were made and found to be essentially the same as reported in November, 1940. Patient had some headache that is controlled by the administration of anterior pituitary extract. Vision at that time was 20/30 right, and 20/15 left.

CONCLUSIONS.

1. The implantation of radium into the pituitary fossa is practical and without danger if the dose is confined to a maximum of 1,000 mgm. hours. This can be accomplished without damage to the optic nerves and surrounding brain structures.

2. In none of the five cases was there any postoperative infection. This can be accounted for on the basis of strict

asepsis and the limiting membrane — the diaphragma sellae.

3. The application of radiation together with the decompression of the tumor explains the elimination of the danger of secondary swelling often accompanying X-ray therapy that necessitates a decompression to save vision.

4. The above procedure still leaves the pituitary fossa accessible by the transfrontal route and amenable to further deep X-ray therapy if necessary, inasmuch as the decompression of the tumor has already been accomplished.

5. Being entirely aware that in such a small series of cases it is impossible to draw definite conclusions regarding the ultimate outcome of this particular treatment, I may say in closing that my only definite finding is the establishment of a safe dosage of radium in this area.

BIBLIOGRAPHY.

RIDGES, A. J.: *Trans. Pacific Coast Oto-Ophthalmol.* Soc., 1931.

CUTLER, MAX, and BUSCHKE, FRANZ: Cancer, Its Diagnosis and Treatment. W. B. Saunders Co., 1939.

MARIE, P.: L'acromegalie. *Progres med.*, Series 2, 9, 189, 1889.

BABINSKI, J.: Tumeur du corps pituitaire sans akromegalie et avec arret de developpement des organes genitaux. *Rev. Neurol.*, 8:531, 1900.

FROHLICH, A.: Ein Fall von Tumor der Hypophysis cerebri ohne akromegalie. *Wien. Klin. Rundschau*, 15:883, 1901.

CUSHING, H.: The Basophil Adenomas of the Pituitary Body and Their Clinical Manifestations (Pituitary Basophilism). *Bull. Johns Hopkins Hosp.*, 50:137, 1932.

SCHLOFFER, H.: Zur Frage der Operationen an der Hypophyse. *Beitr. z. klin. Chir.*, 1:767, 1906.

KANAVEL, A. B.: The Removal of Tumors of the Pituitary Body by an Intranasal Route. *Jour. A. M. A.*, 53:1704, 1909.

HALSTEAD, A. E.: The Operative Treatment of Tumors of the Hypophysis. *Surg., Gynec. ond Obstet.*, 10:494, 1910.

HIRSCH, O.: Uber Methoden der operativen Behandlung von Hypophysistumoren auf endorasalem Wege. *Arch. f. Laryngol. u. Rhinol.*, 24:129, 1910.

CUSHING, H.: The Pituitary Body and Its Disorders. J. B. Lippincott Co., Philadelphia, 1912.

CUSHING, H.: Surgical Experiences with Pituitary Disorders. *Jour. A. M. A.*, 63:1515, 1914.

CUSHING, H.: Les syndromes hypophysaires au point de vue chirurgical. *Rev. Neurol.*, 38, 779, 1922.

KRAUSE, F.: Freilegung der Hypophysis. *Deutche Klin.*, Berl. and Wien., 8:1004, 1909.

FRAZIER, C. H.: An Approach to the Hypophysis Through the Anterior Cranial Fossa. *Ann. Surg.*, 59:145, 1913.

HEUER, G.: Surgical Experiences with an Intracranial Approach to Chiasmal Lesions. *Arch. Surg.*, 1:368, 1920.

DYKE, C. G., and CROSS, S. W.: Roentgenotherapy of Pituitary Tumors. *Bull. Neurol. Inst. N. Y.*, 1:211, 1931.

FRAZIER, C. H.: Surgery of the Pituitary Lesion. *Ann. Surg.*, 88:1, 1928.

CUSHING, H.: Intracranial Tumors. C. C. Thomas, Springfield, Ill., 1932.

DANDY, W. E.: Practice of Surgery (Dean Lewis), 12, 1932. W. F. Prior Co., Inc., Hagerstown, Md.

BECLERE, A.: Le traitement medical des tumeurs hypophysaires, du gigantisme et de l'acromegalie par la radiotherapie. *Bull. et mem. Soc. med. d. hop. de Paris*, 3 series, 27:274-293, 1909.

BECLERE, A.: La radiotherapie des tumeurs de l'encephale. *Rev. Neurol.*, 2:381-382, 1931.

CUSHING, H.: Basophil Adenomas of Pituitary Body and Their Clinical Manifestations. *Bull. Johns Hopkins Hosp.*, 50:137-195, 1932.

CUSHING, H.: Further Notes on Pituitary Basophilism. *Jour. A. M. A.*, 99:281-284, 1932.

CUSHING, H., and WALKER, C. B.: Chiasmal Lesions with Especial Reference to Homonymous Hemianopsia with Hypophyseal Tumor. *Arch. Ophthalmol.*, 47:119-145, 1918.

DOTT, N. M., and BAILEY, PERCIVAL: Consideration of Hypophysial Adenomata. *Brit. Jour. Surg.*, 13:314-365, 1925-1926.

GRAMEGNA, A.: Un cas d'acromegalie traite par la radiotherapie. *Rev. Neurol.*, 17:15-17, 1909.

KUPFERLE, L., and SZILY, A. V.: Die Strahlenbehandlung der Hypophysentumoren. *Lehrb. d. Strahlenterapie* (Gudzent-Holthusen), 3:267-322, 1926.

OTOSCLEROSIS.*

DR. HANS BRUNNER, Chicago.

Even in recent textbooks on otology there are clinical as well as pathological statements that do not agree with the observations of the writer; therefore, the main purpose of this paper is to present my experiences over a period of more than 20 years.

Inasmuch as the clinical diagnosis of otosclerosis is frequently somewhat doubtful, only 17 instances which were studied clinically as well as pathologically are reported. For reasons to be explained later, one case is added which was examined only clinically.

It is not in the scope of this paper to quote the literature to a full extent, since until 1929, the monograph "Otosclerosis" published by the American Otological Society, and the paper of J. G. Wilson,[7] "Present Status of the Problem of Otosclerosis," present an exhaustive review of the work of different investigators.

OTOSCLEROSIS AND SEX.

It is a common statement that otosclerosis affects women more frequently than men; this statement is not entirely correct. If one considers clinical statistics the preponderance of women is obvious. Nager[4] estimates the ratio of otosclerosis in male and female as 1:1.8, and this approximately agrees with the experience of other otologists, as well as that of the writer. The ratio of the sexes is quite different if based on the pathologic findings. I have examined the temporal bones of 17 otosclerotics, nine men and eight women. Lange[3] examined the temporal bones of 40 patients, comprising 26 men and 14 women. Similar results were obtained by Nager and Meyer,[5] who examined the temporal bones of 41 patients, 21 men and 20 women. If the three statistics are combined, the ratio of men to women is 1:0.75. Thus the clinical and path-

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ological statistics furnish contrary results, which points to the conclusion that it is not the otosclerosis *per se* but the clinical progress of otosclerosis which is sex-linked.

DEAFNESS.

The chief symptoms of otosclerosis are bilateral deafness and tinnitus. The deafness may be due *a.* to an ankylosis of the stapes, *b.* to a partial obliteration of the holes of the tractus foraminosus by otosclerotic bone, and *c.* to a congenital malformation of the cochlea including an underdevelopment of the spiral nerves and ganglia. Inasmuch as otosclerosis is never a cause of death, the pathological statistics do not offer a proper view concerning the frequency of deafness in otosclerosis. Among the 17 cases presented there was diminished hearing in 12 cases (eight women and four men), providing the preponderance of women as far as deafness due to otosclerosis is concerned. No audiometric examinations were made in these cases, but in the majority, tuning forks were used. Among the 12 cases presenting marked diminution of hearing, at autopsy eight cases showed chronic diseases; *i.e.*, syphilis of the blood vessels, chronic otitis media, chronic middle ear catarrh, brain tumors, severe arteriosclerosis, any of which might impair the hearing without intercurrent otosclerosis. In the remaining four cases, there were two women with stapes ankylosis; one with a severe congenital malformation of the cochlea, but without ankylosis of stapes, and one woman, 51 years of age, with a simple atrophy of cochlea but without ankylosis of the stapes.

ANKYLOSIS OF STAPES.

Although my very small statistics prove that otosclerosis might lead to a marked diminution of hearing without producing ankylosis of the stapes and without concurrent chronic diseases such as lues, arteriosclerosis, etc., there can be no doubt that among the causes of deafness in otosclerosis, ankylosis of the stapes is of paramount importance. Nager and Meyer[5] found it in 41 per cent; I found it in only 17.8 per cent of microscopically proven cases. I believe, however, that even the figures presented by Nager and Meyer are too low. Usually a diagnosis of ankylosis of the stapes will be made if the otosclerotic bone occupying the frame of the window pene-

trates the annual ligament and extends into the stapes. If one considers that a microscopic specimen through the temporal bone has an average thickness of 0.02 mm. and that usually not every section is stained and mounted, it is obvious that a stapes ankylosis of small extent can be overlooked. Furthermore, in otosclerosis without ankylosis of the stapes there are frequently calcium deposits in the annular ligament. These findings, although not presenting a typical ankylosis of the stapes, are sufficient to impair the motility of the stapes, inasmuch as the stapediovestibular syndesmosis is a very delicate and complicated structure, as proved by the findings of Brunner and Schnierer.[2] These are the reasons for considering ankylosis of the stapes as more frequent as proved by the pathologic statistics. Notwithstanding this conclusion, one must remember that otosclerosis not always results in ankylosis of stapes and that every stapes ankylosis is not due to otosclerosis.

UNILATERAL OTOSCLEROSIS.

As far as the bilateral affection is concerned, otosclerosis as a rule is a bilateral disease. Nager and Meyer[5] found unilateral involvement in 15 per cent of the cases. Among the 17 cases presented, in four instances only one temporal bone was examined. In the remaining 13 there were no less than four with unilateral affection; in all of these cases the otosclerosis was of the latent type; *viz.*, not producing clinical symptoms. The existence of unilateral otosclerosis, therefore, is definite.

TINNITUS.

Every otologist is familiar with the importance of tinnitus as a symptom in otosclerosis. Among the 17 cases presented, in five instances we cannot say whether or not the patients suffered from tinnitus. In the remainder of the cases (12) there were 11 cases without tinnitus and only one in which tinnitus was a predominant symptom. It is, of course, probable that several of these patients had tinnitus in previous years and had simply forgotten it at the time of examination. Among these 11 cases there were four patients who died at the age of 20 to 40 years; *viz.*, an age at which tinnitus due to otosclerosis usually reaches its climax.

Further analysis of these 11 cases without predominant tinnitus proves that there was one instance of ankylosis of

the stapes (a woman, 51 years of age), five cases of atrophy of the cochlea and five cases showed a marked diminution of hearing from different reasons. The number of cases is too small to draw definite conclusions; nevertheless, it seems that neither ankylosis of the stapes nor atrophy of the cochlea necessarily produces tinnitus, at least not permanent tinnitus. Since our knowledge of the pathology of tinnitus is extremely limited, further study of this question is necessary.

PREGNANCY AND OTOSCLEROSIS.

The question concerning the influence of pregnancy upon the progress of deafness in otosclerosis has not yet been uniformly answered. A number of otologists strictly advise against pregnancy in every case of otosclerosis. Some otologists even doubt the correctness of the diagnosis "otosclerosis" if pregnancy did not affect the hearing. There is, of course, no doubt that pregnancy frequently does impair the hearing; however, it is not a hard and fast rule. There are cases of otosclerosis which are not markedly impaired even by several pregnancies and there are cases of otosclerosis in which the hearing diminishes with rapidity without pregnancy. The following cases tend to prove these statements:

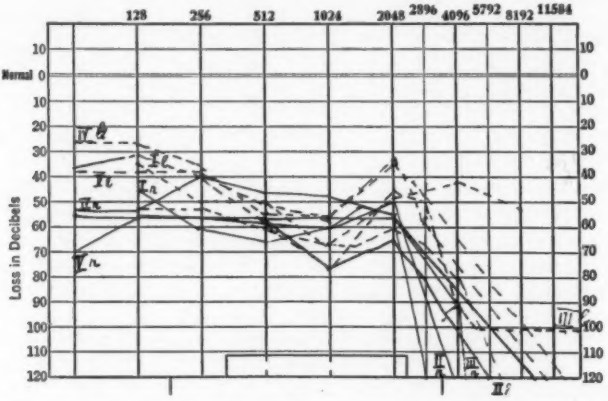
Case 1: A woman 36 years old, died from pneumonia and a cerebellopontile angle tumor on the left side. Microscopic examination of the temporal bones revealed otosclerosis at the tip of the right cochlea but no otosclerosis on the left side. This woman was frequently examined, although never by means of the audiometer. Her hearing for whispered voice on the left side, viz., on the side of the cerebellopontile angle tumor, was always normal, while on the right side, viz., on the side of the otosclerosis, it fluctuated between 1½ to 12 meters, the fluctuation being due to a chronic middle ear catarrh and a stagnation hydrops of the internal ear. Rinne's test changed between positive and negative on both sides; her hearing for high as well as for low tones was normal on both sides in the majority of examinations, as was her bone conduction. Weber's test was always lateralized to the side of the otosclerosis. Two findings are important in this case: *1.* she never complained of tinnitus; *2.* she gave birth to three children and had one abortion without impairment of hearing, although her cerebellar symptoms due to the brain tumor were definitely increased during the pregnancies.

Case 2: A woman, 51 years old, died from a meningitis originating from a chronic suppuration in the region of the right Eustachian tube. The microscopic examination of the temporal bones revealed bilateral otosclerosis in the area of the oval window leading to an ankylosis of the stapes. This woman was hard-of-hearing, particularly on the right side, for many years prior to her death; however, she heard speech. Ten years prior to her death she was involved in an automobile accident and sustained a head injury but no fracture of the skull. Immediately after the injury she became very deaf and heard words only if shouted into

her ears. It is noteworthy that this patient never complained of tinnitus and was never pregnant.

In the first case four pregnancies had no influence upon the otosclerosis neither from the clinical nor from the pathological point of view; while the second patient became very deaf without interfering pregnancy. A great number of clinical observations also prove the uncertainty of the relation of pregnancy to otosclerosis. The following case is of particular interest:

Case 3: Mrs. F. C., 35 years of age, white. Twelve years ago she noticed a diminution of hearing. She does not know whether or not she had



tinnitus at that time, but tinnitus did appear later. There is no deafness in her family. Gradually her deafness grew worse and three years ago a bilateral fenestration operation was performed. Following the operation the tinnitus improved but it is still present. As far as the hearing is concerned the patient believes that the operation was a failure on the left side, but states that there is a permanent improvement of 1 per cent on the right side. As the audiograms made prior to the operation are not available, her statements cannot be checked. She complains, however, of a diminution of her sense of balance since the operation.

In 1940 she gave birth to a child, the birth being instrumental. This pregnancy had no influence upon either her hearing or her tinnitus. Curve I, Fig. 1, shows the audiogram made Oct. 15, 1940, about one year after the fenestration operation and several months after the first birth. In 1932 she gave birth to a second child, the birth being spontaneous. She states that after the last pregnancy her hearing became worse, while the tinnitus was not changed. Curve II, Fig. 1, is the audiogram made Sept. 8, 1942, about three years after the operation and several months after the second birth. Comparison with Curve I does not prove a definite impairment of hearing as stated by the patient.

I saw the patient for the first time on Oct. 27, 1942. An endaural fenestration had been performed on both sides. The external canal was short and straight, so that the drum and the mastoid antrum were exposed to

the air. She wore a hearing aid on the right side. The right antrum was largely opened toward the external canal and entirely epithelialized. The drum was intact. There was a marked scaling excema in the external canal and in the antrum. There was a typical fistula symptom on both sides.

The left ear was filled with scales and dry debris. It was very difficult to remove this debris as every movement caused a severe nystagmus to the left due to an active fistula in the labyrinth. The nystagmus bothered the patient more than the accompanying dizziness to which she was accustomed. After very careful manipulation it was possible to remove the scales and the debris and then the ear presented the same findings as the right ear. Curve III was made Oct. 27, 1942.

The patient inquired whether there was any objection to her having a third child and she was advised against it. The reason was that she had two children and that it was not advisable to risk further impairment of her hearing and eventual abnormality of the child. Despite that reasoning she became pregnant, and Curve IV, taken on April 6, 1943, presents her hearing in the fourth month of her third pregnancy. There was no impairment of her hearing; on the contrary, her hearing was even a little improved. Several weeks after the examination she had a miscarriage, and Curve V, taken on July 13, 1943, presents her hearing two and one-half months after the miscarriage, proving that the pregnancy and the miscarriage had no definite influence upon her hearing.

I believe that every otologist has observed patients who experienced a marked diminution of hearing during or after pregnancy, and some who did not. It seems quite incidental whether one has observed more cases of the one or the other type. Alexander and Stein[6] came to the following conclusions concerning their own cases:

During First Pregnancy		2nd Pregnancy	3rd and More Pregnancies
Otosclerosis becomes manifest	5% (31.2%)	10% (12.5%)	5% (15.6%)
Otosclerosis permanently impaired	30% (31.2%)	60% (9%)	80%
Otosclerosis transitorily impaired	15%	20%	10%
Otosclerosis not impaired	50% (3%)	10%	5%

The figures shown in parentheses are the figures given by Stein. Although the number of instances observed by each author is not great, the difference of the figures is so conspicuous that it cannot be explained by the small number alone, inasmuch as clinical experience of other otologists leads to the same difference of opinion.

I believe that up to the present the problem has not been analyzed properly. The term "pregnancy" includes so many features which eventually can damage the ear that it must be stated, at first, which symptoms of pregnancy do or do not affect the hearing. Perhaps the problem might be stated precisely that way: Either otosclerosis is not influenced by the essential and constant features of pregnancy such as enlargement of uterus, nourishment of embryo, etc., but

rather is influenced by the inconstant and accessory features of pregnancy such as endocrine, vasomotory or psychical disturbances; or otosclerosis is influenced by the essential and constant features of pregnancy, but there are different types of otosclerosis which do or do not respond to these features. Each possibility may occur, but up to the present neither has been proven. Under these circumstances it is better to avoid general statements concerning the relation of pregnancy and otosclerosis and to handle every case individually. Frequently the findings of otosclerosis will be of less importance than other factors, such as heredity which concerns not only the ear but also mental and endocrine diseases. Furthermore, the question of whether or not a woman has children may depend upon her age, general health (particularly the status of her autonomous nervous system and the balance or imbalance of her endocrine glands), social circumstances and other factors.

REFERENCES.

1. Alexander, G.: Die Schaedigung des Gehororganes durch Geschlechtsfunktion und Mutterschaft. *Monatssch. f. Ohrenheilk.*, 1, 1925.
2. Brunner, H., and Schnierer, J.: *Collegium Otolaryngol.*, 134, 1937.
3. Lange, W.: Ueber die Anfaenge der otosklerotischen Herderkrankung. *Ztschr. f. Hals., Nasen. u. Ohrenheilk.*, 25:1, 1930.
4. Nager, F. R.: Zur Klinik und pathologischen Anatomie der Otosklerose. *Acta Otolaryngol.*, 27:542, 1939.
5. Nager, F. R., and Meyer: Die Erkrankungen des Knochensystemes und ihre Erscheinungen an der Innenohrkapsel des Menschen. Berlin, 1932, Karger.
6. Stein, C. Graviditaet und Otosklerose. *Monatssch. f. Ohrenheilk.*, 511, 1925.
7. Wilson, J. G.: Present Status of the Problem of Otosclerosis. *Arch.* Otolaryngol., 28:946, 1938.

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MENIERE'S DISEASE. SURGICAL APPROACH TO THE LABYRINTH WITH DESTRUCTION OF THE UTRICLE; CASE REPORTS.*

DR. HENRY M. GOODYEAR, Cincinnati.

In this paper is considered only the limited number of cases which do not respond to medical treatment or the removal of foci of infection, and in which, apparently, relief from symptoms can be obtained only by destruction of the function of the labyrinth.

Hallpike and Cairns, in 1938, described the pathological findings of the postmortem examinations performed on two patients who had suffered from Ménière's syndrome. In both cases gross dilatation of the saccule and scala media with obliteration of the perilymph space and of the scala vestibuli had occurred.

Once the endolymph system has attained its maximal degree of dilatation, an infinitesimal quantity of additional endolymph fluid against the very sensitive membranous labyrinth would produce abnormal labyrinth stimulation.

Considering the possibility of this theory as expounded by Hallpike, I reasoned that a decompression of the membranous labyrinth should give relief, and, more than four years ago, in May, 1939, did such an operation on a woman, aged 40, who had severe attacks of dizziness, with tinnitus in the left ear and impaired hearing in both ears, but worse in the left. Salt-free diet and potassium chloride had given only temporary relief and the attacks were growing more frequent and severe. One week before operation while ironing she was suddenly seized with an attack and fell to the floor. Tinnitus in the left ear was always worse during and following each attack.

Operation; Case 1: Under local anesthesia a postauricular incision was made and the mastoid cortex opened with a trephine and burr. The hori-

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zontal and superior semicircular canals were carefully exposed along with the short limb of the incus. The bone between the ampulla of the horizontal and superior semicircular canals was removed to expose the membranous labyrinth and the removal of bone over the vestibule was carried as far forward as possible.

The decompression was then covered with a skin flap as I have described in the fenestration operation.[2]

Two days after operation two severe attacks of Ménière's occurred and a third attack five months later. Since that time in a matter of four years there have been only occasional attacks of dizziness, which have been mild in character, not requiring further treatment.

The hearing completely disappeared after four months, but the patient today is extremely sensitive to the fistula test of air compression and suction.

Since the labyrinth was still sensitive in this case, with occasional mild dizziness, I decided to remove or destroy the utricle in the next case which came to me for operation.

Case 2: A colored woman, aged 42, who complained of severe attacks of dizziness with nausea, loss of hearing, and tinnitus in the right ear. Attacks were occurring two to three times per week and intermittent attacks had occurred for three years. There was an 80 per cent loss of hearing in the right ear and a 50 per cent loss in the left.

Under local anesthesia an exposure was made as in Case 1. With a small electric burr the bone between the horizontal and superior ampulla was removed and the vestibule exposed anteriorly to permit the entrance of a small dental excavator which was used to destroy the utricle well down toward the ampullae of the posterior semicircular canal. The wound was completely closed with silk without a drain.

There was a quick nystagmus to the left with nausea, but on the third day the patient was out of bed and walked without support on the fourth day.

For seven days after operation there was apparently little decline in hearing in the ear operated upon, tested with a Barany noise apparatus applied to the opposite ear; however, by the tenth day the hearing in the right ear completely disappeared, while tinnitus remained.

The patient has been free from any sudden attacks of dizziness since operation and is much pleased with the result.

Case 3: A white, unmarried woman, aged 25, who had her first attack of severe dizziness 18 months previous. There was deafness and tinnitus in the left ear. There was nausea with each attack and sometimes vomiting. Two weeks before operation she fell to the sidewalk on her way to work, lacerating the scalp, and was unconscious.

On the neurological service at the Cincinnati General Hospital attempts were made to reproduce the attacks by the use of enteric coated NaCl without avail.

The spinal fluid was normal and negative for Wassermann reaction. There was a 50 per cent loss of hearing in the left ear with normal hearing in the right.

Operation as in Case 2 was performed under local anesthesia.

The patient was up on the third day and left the hospital on the fifth.

There was no hearing in the left ear when tested with a Barany noise apparatus in the right on the twelfth day.

It is now one year since the operation and the patient states she has not had an attack of dizziness since the operation.

Case 4: A male aged 35, whose first attack of severe dizziness occurred 15 months previous while he was sitting in his office. The attack came without warning and he vomited for one hour. There was tinnitus in the right ear. In the 15 months following he had eight violent attacks and many of minor severity. The hearing was normal in the left ear but showed a 25 per cent loss in the right.

Excellent medical treatment had been given and consultation had with a neurologist and neurosurgeon; both agreed it was a true case of Ménière's.

Operation was performed as in Cases 2 and 3. Four months have elapsed and the patient has been free from symptoms of Ménière's syndrome. He is back in his executive position, has gained weight and is very happy to be free of constant fear of falling.

There is some tinnitus present and the hearing is completely gone in the ear operated upon.

SUMMARY.

It was evident that the decompression of the labyrinth was helpful in the first case but the relief was not complete. In the other three cases where the utricle, which anatomically contains neuroepithelium, was destroyed or removed, the function of the labyrinth was completely eliminated.

I feel that the postauricular approach is preferable for this particular lesion and in all my cases great care was used not to disturb or injure the posterior external auditory canal wall. To open this wall would add the danger of possible secondary infection from the skin of the external auditory canal.

In all my cases there was no painful disturbance of the middle ear or rupture of the tympanum following operation, and all cases healed by first intention.

By using the trephine and burr the operation was done with ease under local anesthesia. One and one-half grains of nembutal were given, followed one-half hour later by one-fourth grain of morphine and one one-fiftieth of scopolamine one hour and fifteen minutes before operation. A 1 per cent solution of novocaine was injected into the skin and periosteum at operation.

In the last three cases each operation was completed in less than one hour with deliberation.

In all three cases where the utricle was destroyed, there

resulted a complete loss of hearing; however, Dandy reports that the hearing is of little value in late cases of Ménière's syndrome; however, in my next case I expect to limit the destruction to the ampullae of the superior and horizontal canals and possibly extend the operation to include only the otolithic membrane which is in the immediate area and is most prominent at the opening of the horizontal canal. I am hoping that the operation may be modified so that such hearing as is present may remain.

REFERENCES.

1. HALLPIKE, C. S., and CAIRNS, H.: *Proc. Roy. Soc. Med.*, p. 1317, Sept., 1938.

2. GOODYEAR, HENRY M.: Otosclerosis, Postauricular Approach in Operative Treatment. *Arch. Otolaryngol.*, 31:451-457, March, 1940.

3. DANDY, W. E.: The Surgical Treatment of Ménière's Disease. *Surg., Gynec. and Obstet.*, 72:421, 1941.

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UNIVERSITY OF MINNESOTA MEDICAL SCHOOL CONTINUATION COURSE IN OTOLARYNGOLOGY.

The University of Minnesota Medical School, Division of Otolaryngology, announces its bi-annual Continuation Course in Otolaryngology, Feb. 7-11, 1944. This course is clinical and registration is limited to 50 physicians practicing otolaryngology as a specialty. The course will be conducted by members of the Graduate School Faculty of the University and the Mayo Foundation. A guest faculty will include Dr. Oscar Batson, of Philadelphia, Dr. Paul Holinger and Dr. John Lindsay, of Chicago, and Dr. Theodore Walsh, of St. Louis. The tuition is $25. Accommodations will be reserved for registrants in a Minnesota hotel because the Center for Continuation Study is now occupied by men in military training. Early registration is desirable. Apply to Dr. A. W. O'Brien, Director Post-Graduate Medical Education, University Hospital, Minneapolis.

HEMATOLOGY.

DR. JOHN J. SHEA, Memphis.

The war has made the world blood conscious. Donations to blood banks have equaled the subscriptions to our war loans. In an appeal to the public for blood contributions a slogan has been formed, "Don't be a turnip." Fowler and Barer[1] reported the rate of hemoglobin regeneration in blood donors to be in the majority of instances less than 50 days, but that 25.8 per cent of them required two months to re-establish their normal averages. Multiple donations increase the time required and is slower in females than in the males. The administration of 1 gm. daily of iron and ammonium citrates will shorten the period of regeneration. Blain and Campbell[2] describe the following method of preparing a solution of oxalic acid for intravenous administration to increase the coagulating ability of the blood. "Since oxalic acid in aqueous solution is unstable at high temperatures, overheating must be avoided in its preparation, and pressure sterilization cannot be utilized. A saturated solution is first prepared by dissolving chemically pure oxalic acid in sterile distilled water at 60° C. This solution is cooled in a refrigerator to 5° C., and the precipitated oxalic acid is filtered. Strict aseptic precautions should be observed in each step. Ten grams of dried recrystallized oxalic acid are then dissolved in 800 cc. of sterile distilled water. One hundred and forty cc. of normal sodium hydroxide are then added, sufficient distilled water being finally added to bring the volume to 1 liter. The hydrogen ion concentration of this solution then varies between 3.5 and 4.3. The solution is next filtered through a Bakefeld filter and then the finished product is placed in sterile amber ampules or bottles of a capacity not greater than 5 cc. These ampules or bottles are stored in a refrigerator. A small amount of phenol may be added as a preservative if desired. One cc. of the finished product contains 10 mg. of oxalic acid."

BLOOD TRANSFUSIONS.

In spite of blood grouping and cross-matching, reactions

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follow the administration of whole or citrated blood. In the beginning this was blamed upon the A and B agglutinogens, but Landsteiner and Wiener[3] described a new factor called by them the Rh agglutinogen. This factor was prepared by immunizing rabbit against rhesus monkey blood and is present in about 85 per cent of individuals tested. If the 15 per cent of Rh negative patients have repeated transfusions of blood from Rh positive patients, they will produce an antibody capable in future transfusions of causing anaphylaxis. This condition is exaggerated in postpartum transfusions if the wife is a Rh negative individual and the husband as donor is Rh positive. Most likely the offspring would have been a Rh positive and the mother due to her contact with the fetus would have developed anti-Rh bodies.[4,5,6] A further progress of this condition has a tendency in some women to have all except her first infant to develop erythroblastosis.[7]

Rucks and Hobson[8] reported a case with recovery of "Purpura Fulminans (Waterhouse Friderichsen Syndrome)." To the otolaryngologist this blood stream infection is a rare condition and frequently fatal but should be considered when focal infection is studied as the cause of purpura.

Calder and Kerby[9] state that the addition in vitro of 0.6 mgm. of nicotinic acid to 1 cc. of blood reduced the clotting time in six brucellosis patients from 11.3 to 6 minutes and the clot retraction was completed within three hours. The clotting time of plasma from 25 brucellosis patients was likewise reduced by about 36 per cent. These observations were extended to a small group of patients with other clinical states associated with bleeding (typhoid fever, prolonged nose bleeds, hypoplastic anemia, low grade fever of unknown etiology with a tendency to bleed, acute catarrhal jaundice, bleeding subsequent to tooth extraction) and the results were uniformly encouraging. Presumably the hemorrhagic tendency in these cases rested on a toxic, infectious basis. The authors have not yet tried nicotinic acid in the various blood dyscrasias in which its possible effect would be of obvious interest and importance. In vitro it had no effect on blood from a patient with hemophilia. Experimental studies show that nicotinic acid does not duplicate the action of, and cannot be substituted for, any of the known factors involved in blood clotting (calcium, platelets, thromboplastic principle,

prothrombin or thrombin); however, it does induce coagulation when added in vitro to blood containing excessive amounts of antithrombin (heparinized blood or blood from dogs in peptone or anaphylactic shock). Contrast with seven other acids, none of which was as active in this respect, indicates that this effect is not due to nonspecific alteration of hydrogen-ion concentration.

SULFANILAMIDE AND ITS DERIVATIVES.

Hemolytic Anemia: Sulfonamide compounds may produce an anemia after the second day of administration but usually require two weeks to effect an appreciable reduction in the number of the white corpuscles. Trier[10] analyzed 90 cases of anemia the result of chemotherapy and reported three fatalities. The anemia has a sudden onset with a rapid reduction of the number of red corpuscles and the hemoglobin value. Hendricks[11] reported that 40 per cent of 433 patients showed an anemia following the administration of these drugs and that infants and children were more susceptible than adults.

GRANULOCYTOPENIA AND AGRANULOCYTOSIS.

When either of these conditions occurs, the individual is sensitive to one or a combination of these drugs, and there occurs a partial paralysis of the hematopoietic organs. The condition is rare and usually follows two weeks of therapy. Many authorities suggest the use of chemotherapy when the agranulocytosis is the result of an overwhelming infection. Jackson[12] failed to separate the true agranulocytosis from those the result of drugs or chemicals. It is an accepted opinion that the septic cases retain at least 10 per cent of the leucocytes as polynuclears. The accepted therapy for these conditions remain pentose nucleotides, liver extracts, transfused blood and yellow bone marrow extract, and when sepsis predominates, the judicial administration of the sulfonamide compounds.

INFECTIOUS MONONUCLEOSIS.

Nettleship[13] discusses the bacteriology of the disease. McFarland[14] regrets the failure of many physicians to diagnose mononucleosis and Ryan[15] stresses the careful study of the blood to isolate the atypical lymphocytes and emphasizes the

necessity of repeated heterophile tests. Ten cases of conjunctivitis have been reported by Ash and Arbogast.[16] The symptoms of acute appendicitis have been reported by Straus[17] and others, the result of lesions in the lymphoid tissues of the appendix. Seeds[18] emphasizes the necessity of considering infectious mononucleosis in children with extensive cervical adenitis.

Several excellent articles[19,20] have appeared dealing with the heterophile antibody test emphasizing that transient false positive serologic reaction for syphilis may occur.

Straus and Bernstein[21] "have made an investigation of the five different techniques for the demonstration of the sheep cell agglutinins in the blood serum of patients with infectious mononucleosis. According to them, each of the following factors has an important effect on the sensitivity of the test: the amount of blood serum employed; the serial dilution of the serum; the concentration of the sheep cell suspension added to the serum; the temperature at which the mixtures are maintained. After a careful serologic study of a large number of patients with and without infectious mononucleosis, they conclude the series determinations are of more diagnostic value than single determinations and that the "single tube test," introduced by Straus in 1936, is of diagnostic value when positive but does not exclude the disease when negative. They state that increased titers of heterophile agglutinins had been reported for the blood serums of patients with serum sickness, pneumonia, scarlet fever, measles, tuberculosis, filariasis and aplastic anemia, and for those patients treated with quinine or parenteral liver extract."

Treatment: It remains the same symptomatic therapy with potassium bismuth tartrate being the nearest to a specific. Berkley[22] administered 100 cc. of scarlet fever convalescent serum to four patients with brilliant results. Smyth[23] recorded a prompt recovery following the daily administration of four doses of 20 cc. of serum from a patient convalescing from mononucleosis. The use of the potassium salt of bismuth tartrate rather than the sodium is appreciated by the patient as it is more soluble and the local reaction of soreness is less. Two cubic centimeters of bismuth and potassium tartrate 0.03 gm. (Lilly) are injected intramuscular daily for

three doses, and then every other day until relief is obtained. If the condition proves to be a simple tonsillitis, the drug is ideal.

HODGKIN'S DISEASE.

Slaughter and Craver[24] reviewed a series of 265 cases of Hodgkin's disease at the Memorial Hospital, New York. This paper is worthy of complete study because of the increased interest in nasopharyngeal tumors. Two methods of Roentgen therapy are described, palliative and obliterative, besides a suggestion of the value of early surgical removal of the lymph nodes.

LEUCEMIA.

The oral lesions of leucemia have been reported in several of the dental journals in which simple toothache[25] was the earliest symptom of the blood dyscrasia, while in others gingival hypertrophy[26,27] with bleeding gums was the initial manifestation. Unfortunately, in otolaryngologic literature there was no report of any tonsillar lesion, although this condition is fairly frequent. The addition of radioactive phosphorus (P_{32}) therapy in the treatment of leucemia is still in the experimental stage and is used instead of local radiation. Craver[28] reports that the radioactive phosphorus is absorbed by cancerous tissue allowing irradiation of the malignant cells by the spray method with a minimum damage to the normal tissues. Two oral methods of administration are described by Low-Beer, Lawrence and Stone.[29] The first consists in the irregular administration of 6 to 10 millicuries and the second, frequent small doses 0.5 to 3 millicuries every three days.

Bompet and Montero[30] in an article on "Ascorbic Acid Therapy in Otorhinolaryngology" voice the opinion that ascorbic acid influences the calcium metabolism and improves the hemostyptic function, increases thrombocytopoeisis and activates the thrombin. The accepted idea in this country is that the part played by vitamin C in coagulation is its action upon the capillaries.

BIBLIOGRAPHY.

1. Fowler, W. M., and Barer, A. P.: Rate of Hemoglobin Regeneration in Blood Donors. *Jour. A. M. A.*, 118:421, Feb. 7, 1942.

2. Blain and Campbell: *Arch. Surg.*, 44:1117, June, 1942.

3. LANDSTEINER, KARL, and WIENER, ALEXANDER S.: Studies on an Agglutinogen (Rh) in Human Blood Reacting with Anti-Rhesus Sera and with Human Isoantibodies. *Jour. Exper. Med.*, 7:309, Oct. 1, 1941.

4. DIAMOND, LOUIS K.: Hemolytic Transfusion Reaction Due to the Rh Factor: A Preventable Danger. *N. Eng. Jour. Med.*, 227:857, Dec. 3, 1942.

5. FISK, ROY T., and FOORD, ALVIN G.: Observations on the Rh Agglutinogen of Human Blood. *Am. Jour. Clin. Path.*, 12:545, Nov., 1942.

6. DAVIDSOHN, I., and TOHARSKY, B.: The Rh Blood Factor; An Antigenic Analysis. *Am. Jour. Clin. Path.*, 12:434, Aug., 1942.

7. LEVINE, PHILIP: The Pathogenesis of Fetal Erythroblastosis. *N. Y. State Jour. Med.*, 42:1928, Oct., 1942.

8. RUCKS, W. L., and HOBSON, J. J.: Purpura Fulminans (Waterhouse-Friderichsen Syndrome). *Jour Pediat.*, 22:2:226-232, Feb., 1943.

9. CALDER, R. M., and KERBY, B. P.: The Effect of Nicotinic Acid on Blood Coagulation. *Am. Jour. Med. Sci.*, 200:590-596, Nov., 1940.

10. TRIER, M.: Acute Hemolytic Anemia Developing During Therapy with Substances Belonging to the Sulfanilamide Group: Survey and Report of Case. *Acta. Med. Scandinav.*, 108:117, 1941.

11. HENDRICKS, C. B.: Sulfonamide Compounds: Blood Changes Therefrom. *Cal. and West. Med.*, 56:253, 1942.

12. JACKSON, H., JR.: Medical Progress: Leukemia; Agranulocytosis. *N. Eng. Jour. Med.*, 225:978, 1941.

13. NETTLESHIP, A.: On Infectious Mononucleosis. *Proc. Soc. Exper. Biol. and Med.*, 49:116, 1942.

14. MCFARLAND, J. E.: The Clinical Problem of Infectious Mononucleosis. *Jour. Iowa Med. Soc.*, 32:10, 1942.

15. RYAN, J. M.: Infectious Mononucleosis. *Minn. Med.*, 25:871, 1942.

16. ASH, H. H., and ARBOGAST, J. L.: Infectious Mononucleosis. *Jour. Ind. Med. Assn.*, 35:562, 1942.

17. STRAUS, R.: Infectious Mononucleosis Stimulating Acute Appendicitis with Description of a Specific Lesion of the Appendix. *Am. Jour. Clin. Path.*, 12:295, 1942.

18. SEEDS, A.: Acute Cervical Adenitis in Children. *Tex. State Jour. Med.*, 37:592, 1942.

19. LONERGAN, M.: Straus Technique of the Paul-Bunnell Test. *Canad-Med. Jour., Tech.*, 4:107, 1942.

20. CANZANI, R.; VARELA FUENTES, B., and DI BELLO, R.: Falsas positivas de la mononucleosis infecciosa (fiebre ganglionar de Pfeiffer). *Sem. med.*, 2:502, 1941.

21. STRAUS, R., and BERNSTEIN, M. T.: Further Serological Studies in Infectious Mononucleosis. *Am. Jour. Clin. Path.*, 12:174, 1942.

22. BERKLEY, H. K.: Infectious Mononucleosis: Its Treatment with Scarlet Fever Convalescent Serum. *Jour. Pediat.*, 20:26, 1942.

23. SMYTH, L. A.: Report of Case of Infectious Mononucleosis. *Cinn. Jour. Med.*, 23:292, 1942.

24. SLAUGHTER, D. P., and CRAVER, L. F.: Hodgkin's Disease: Five-Year Survival Rate; Value of Early Surgical Treatment; Notes on Four Cases of Long Duration. *Am. Jour. Roentgenol.*, 47:596, 1942.

25. PASTERNACK, J. G.; ABBOTT, G. A., and WERNER, R. D.: Leukemia Gingivopathy: A Case of Acute Lymphatic Aleukemic Leukemia. *Jour. Am. Dent. Assn.*, 29:1193, 1942.

26. ARMBRECHT, E. C., and APPLE, C. A.: Lymphatic Leucemia. *Am. Jour. Orthodon.*, 28:607, 1942.

27. MASON, H.: Gingival Hypertrophy Due to Myelogenous Leucemia. *Am. Jour. Orthodon.*, 28:738, 1942.

28. CRAVER, L. F.: Treatment of Leukemia by Radioactive Phosphorus. *Bull. N. Y. Acad. Med.*, 18:254, 1942.

29. LOW-BEER, B. V. A.; LAWRENCE, J. H., and STONE, R. S.: The Therapeutic Use of Artificially Produced Radioactive Substances: Radiophosphorus, Radiostrontium, Radioiodine, with Special Reference to Leukemia and Allied Diseases. *Radiol.*, 39:573, 1942.

30. BOMPET, RICARDO, and MONTERO, JUAN: La ascorboterapia enotorrinolaringologia. *La Prensa med., argent.*, 29:149-152, Jan. 21, 1942.

THE AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

The Section meetings of the Society are scheduled as follows:

Southern Section—Saturday, Jan. 8—Atlanta.

Middle Section—Wednesday, Jan. 12—Cleveland.

Eastern Section—Friday, Jan. 14—New York.

Western Section—Saturday and Sunday, Jan. 22 and 23—Los Angeles.

The programs, which will be mailed to members in December, contain papers of exceptional merit.

Sentiment in favor of holding the annual meeting has become so strong that preparations for such a session have already commenced.

1. An effort is being made to have the meetings of the American Otological Society, the American Laryngological Association, the American Broncho-Esophagological Association, and the Triological Society held consecutively and in the same city. Chicago and Atlantic City have been suggested as possible meeting places, the time to be in May or June.

2. Dr. Marshall Taylor has a very attractive program nearly completed.

3. Unless unforeseen circumstances prevent, it is expected that the Council, in conjunction with the Councils of other societies, will approve plans for the annual session.

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL THERAPY, AMERICAN MEDICAL ASSOCIATION.

Acousticon Hearing Aid (carbon). *Jour.* A. M. A., 106: 1007, March 21, 1936.

Coronation Acousticon Hearing Aids (carbon). *Jour. A. M. A.*, 111:25, July 2, 1938.

Acousticon Hearing Aids, Gold Medal Models (carbon). *Jour. A. M. A.*, 115:216, July 20, 1940.

Acousticon, Model A-45 (tube). *Jour. A. M. A.*, 117:1978, Dec. 6, 1941. Mfr.: Dictograph Sales Corp., 580 Fifth Avenue, New York.

Aurex Hearing Aid (Semi-Portable) (tube). *Jour. A. M. A.*, 109:585, Aug. 21, 1937.

Aurex Model C-B and Model C-A Hearing Aids (tube). *Jour. A. M. A.*, 120:535, Oct. 17, 1942. Mfr.: Aurex Corp., 1117 N. Franklin Street, Chicago.

Gem Hearing Aid, Model V-4, C-186 (tube). *Jour. A. M. A.*, 120:1397, Dec. 26, 1942. Mfr.: Gem Ear Phone Co., 47 West 34th Street, New York.

Maicophone (tube). *Jour. A. M. A.*, 114:413, Feb. 3, 1940.

Maico Hearing Aid, Model 42 (tube). *Jour. A. M. A.*, 120: 839, Nov. 14, 1942.

Maico Ace Hearing Aid (tube). *Jour. A. M. A.*, 121:48, Jan. 2, 1943. Mfr.: Maico Co., Inc., 2632-36 Nicollet Avenue, Minneapolis, Minn.

Mears Aurophone, No. 98 (tube). *Jour. A. M. A.*, 118:978, March 21, 1942. Mfr.: Mears Radio Hearing Device Corp., 1 West 34th Street, New York.

Otarion (tube). *Jour. A. M. A.*, 115:1101, Sept. 25, 1940. Mfr.: Otarion, Inc., 448 N. Wells Street, Chicago.

Radioear (carbon). *Jour. A. M. A.*, 110:1753, May 21, 1938.

Radioear Masterpiece (carbon). *Jour. A. M. A.*, 117:2169, Dec. 20, 1941.

Radioear Special Electronic Type C Hearing Aid (tube). *Jour. A. M. A.*, 116:2018, May 3, 1941.

Radioear Electronic 41 Junior Hearing Aid (tube). *Jour. A. M. A.*, 116-1645, April 12, 1941.

Radioear Electronic Model 41 Hearing Aid (tube). *Jour. A. M. A.*, 117:291, July 26, 1941. Mfr.: E. A. Myers and Sons, 306-8 Beverly Road, Mt. Lebanon, Pittsburgh.

Ravox Hearing Aid (Semi-Portable) (tube). *Jour. A. M. A.*, 113:1643, Oct. 28, 1939. Mfr.: Zenith Radio Corp., 6001 Dickens Avenue, Chicago.

Telex Hearing Aid (tube). *Jour. A. M. A.*, 114:1634, April 27, 1940.

Telex Hearing Aid, Model 900 (tube). *Jour. A. M. A.*, 117: 1978, Dec. 6, 1941.

Telex Hearing Aid, Model 1020 (tube). *Jour. A. M. A.*. Dec. 13, 1941. Mfr.: Telex Products Co., 1645 Hennepin Avenue, Minneapolis.

Vacolite Vacuum Tube Hearing Aid (tube). *Jour. A. M. A.*, 116:2597, June 7, 1941.

Vacolite Model D Hearing Aid (tube). *Jour. A. M. A.*, 118: 896, March 14, 1942. Mfr.: Vacolite Co., 3003 North Henderson, Dallas.

Western Electric Ortho-tronic Audiphone (tube). *Jour. A. M. A.*, 121:1283, April 17, 1943.

Western Electric Audiphone, Ortho-Technic Model (carbon). *Jour. A. M. A.*, 112:1062, March 18, 1939. Mfr.: Western Electric Co., 300 Central Avenue, Kearny, N. J.

Audiometers Accepted.

Maico Audiometer, Model D-5. *Jour. A. M. A.*, 114:139, Jan. 13, 1940.

Maico Audiometer, Model D-8. *Jour. A. M. A.*, 120:205, Sept. 19, 1942. Mfr.: Maico Co., Inc., 2632-36 Nicollet Avenue, Minneapolis.

Western Electric Audiometer, Model 6B. *Jour. A. M. A.*, 114:1634, April 27, 1940.

Western Electric 4C Audiometer. *Jour. A. M. A.*, 118:1297, April 11, 1942. Mfr.: Western Electric Co., 300 Central Avenue, Kearny, N. J.

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Eastern—Chairman: Dr. Westley M. Hunt, 33 East 68th Street, New York.
Meeting Place: New York, Jan. 14, 1944.

Southern—Chairman: Dr. William C. Warren, Jr., Doctors' Building, Atlanta.
Meeting Place: Atlanta, Jan. 8, 1944.

Middle—Chairman: Dr. Fred W. Dixon, 2060 East 9th Street, Cleveland.
Meeting Place: Cleveland, Jan. 12, 1944.

Western—Chairman: Dr. Simon Jesberg, 500 South Lucas Avenue, Los Angeles.
Meeting Place: Los Angeles, Jan. 22-23, 1944.

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Chairman: Dr. Claude C. Cody, 1304 Walker Ave., Houston.
Secretary: Dr. Louis H. Clerf, 1530 Locust Street, Philadelphia.
Place: San Francisco.

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Secretary-Treasurer: Dr. Orrie H. Ghrist, 210 N. Central, Glendale, Cal.
Committeeman: Dr. Isaac H. Jones, 635 S. Westlake, Los Angeles.
Place: Los Angeles County Medical Association Building, 1935 Wilshire Boulevard, Los Angeles.
Time: 6:00 P.M., fourth Monday of each month, September to May, incl.